BRAIN.

PART IV., 1907.

Original Articles and Clinical Cases.

THE CEREBRAL ARTERIAL SUPPLY.

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Presidential Address delivered before the Neurological Society, February 21, 1907.

FOR the last seven years I have been engaged in working out the distribution of the different arteries to the human brain, and during this time I have injected nearly a hundred brains; but as some of these were necessarily failures, I propose to give you the results of the injection of eighty-seven different brains.

The arteries which I separately injected were the posterior communicating and the anterior choroid—branches of the internal carotid—the anterior, middle, and posterior cerebral arteries. I would here remark that in some text-books the anterior choroid is figured as coming off from the middle cerebral in place of from the internal carotid, and although this may occasionally occur, I do not remember to have met with this distribution in any of my cases.

I must first refer to the work of previous observers, and especially to those of Duret [2], Heubner [3], and Kolisko [4].

As far as I can ascertain, these observers all injected the arteries by means of a syringe, using insoluble particulate

colours or an oil emulsion mass. Duret speaks about using gelatine coloured with carmine, but whether this means soluble carmine I am unable to say, as he does not give any details of the injection he used.

The arterial distribution is divided into basal and cortical, and I will take first the basal supply. The posterior communicating artery, according to Duret, supplies the optic chiasma, the tuber cinereum and pituitary body, the corpus mammillare, two branches to the optic thalamus—especially to the wall of the infundibulum in the most anterior part of the third ventricle and the grey commissure—the optic tract and the cerebral peduncles. According to Heubner this artery supplies the posterior part of the lateral wall of the infundibulum, the inner and anterior part, and sometimes the outer anterior part, of the thalamus opticus, the posterior and the ascending pillars of the fornix, the commissura mollis, the corpus mammillare, and the tegmentum of the crus cerebri. Kolisko makes the posterior communicating artery supply the anterior outer part of the optic thalamus, the anterior quarter of the posterior limb of the internal capsule, the anterior part of the crus cerebri, and sometimes part of the internal segment of the lenticular nucleus.

The anterior choroid artery, according to all observers, supplies the uncinate gyrus and enters the anterior end of the descending cornu of the lateral ventricle, to end in the choroid plexus. On its way to the ventricle it sends in basal branches, which, according to Heubner, supply the highest part of the crus cerebri, the posterior limb of the internal capsule and the anterior external part of the optic thalamus. According to Kolisko these branches supply only the posterior two-thirds of the posterior limb below the level of the second lenticular segment, and, in addition, the white matter behind the internal capsule, the lamina medullaris of the optic thalamus, the internal segment of the lenticular nucleus, the tail of the caudate nucleus, the optic tract, and exceptionally the external superior part of the optic thalamus.

The middle cerebral artery, according to Duret, supplies internal branches to the first and second segments of the

lenticular nucleus and external branches, lenticulo-striate, to the third segment of the lenticular nucleus, and to the tail of the caudate nucleus; and lenticulo-optic branches to the lenticular nucleus and the optic thalamus. Heubner gives the same distribution, but he makes the middle cerebral supply the middle part of the corpus striatum, and branches from the fork between the anterior and middle cerebral arteries supply the first and second segments of the lenticular nucleus and the anterior limb of the internal capsule, while the third or outer segment of the lenticular nucleus is supplied by the middle cerebral. According to Kolisko, the middle cerebral supplies the lenticular nucleus — second and third segments—the upper one-third of the caudate nucleus, and the part of the posterior limb of the internal capsule which lies above the horizontal level of the superior angle of the second lenticular segment.

The anterior cerebral artery, according to all three authors, supplies the head of the caudate nucleus.

The posterior cerebral artery, according to Duret, supplies the crus cerebri, the optic thalamus in its posterior parts, the corpora quadrigemina, the corpora geniculata, the choroid plexus and membrane by the postero-lateral and median choroid arteries, and the cornu ammonis. Heubner, in addition, gives the red nucleus.

With regard to the choroid plexus and membrane, according to Duret the choroid plexus is supplied by the anterior choroid artery in its posterior inferior two-thirds, and by the postero-lateral choroid artery in its anterior superior one-third, *i.e.*, the part above the optic thalamus; the choroid membrane is supplied by the postero-lateral choroid branch of the posterior cerebral artery, while the choroid plexus and membrane of the third ventricle are supplied by the posterior median choroid branch of the posterior cerebral artery. Also, according to Duret, the posterior choroid arteries supply the head of the caudate nucleus, and anastomose with the anterior cerebral artery there.

Heubner also finds that the anterior choroid artery supplies the choroid plexus in the descending cornu of the lateral ventricle, while branches from the posterior cerebral pass to the plexus through the posterior cerebral fissure, and supply

the part of the plexus in the posterior cornu and in the central part of the lateral ventricle.

Taking next the cortical surface, Duret's description is that which is usually adopted, and it is so well known to all that I need not describe it, but later I will point out where my results differ from those of Duret. He also states that the anterior choroid supplies the uncinate convolution, though he does not figure it in his diagrams, and this supply was also found by both Heubner and Kolisko.

Method of Investigation.

The method which I employ, and which, I believe, has not been used before, is to inject simultaneously by means of the same pressure three, four, or five arteries with liquid gelatine containing different soluble colours. The pressure is produced by using two large pressure bottles, connected at their lower openings by a long indiarubber tube, and by raising one of them gradually to from 3 to 6 ft. The other bottle is connected at its upper opening with the upper openings of the three, four, or five smaller pressure bottles, which contain the different coloured gelatines. The smaller bottles are connected at their lower openings by means of fine indiarubber tubes and glass cannulæ with the different arteries, and together with the brain, they are immersed in a basin of water at 40° to 50° C. The brain, after careful removal from the cranial cavity, is injected with water at 50° C. to wash out the blood from the vessels, and it is then injected with gelatine, to which various colours have been added.

After trying, without good results, various oil masses and gelatine containing insoluble powders, I now use entirely soluble colours in gelatine, made in the following way:—

Two ounces by weight [56 grms.] of gelatine are soaked in cold water for half an hour, and then melted in a water bath; to the gelatine the different colours made according to the formulæ given below are added; hot water to make up the quantity to one pint [568 c.c.] is added, and the mass is strained through flannel.

For red, the formula is :—

Carmine	½ drachm [2 grms.].
Ammonia	½ fluid drachm [1·8 c.c.].
Glycerine	½ fluid oz. [14·3 c.c.].

Dissolve and add to the gelatine.

For blue :—

Nicholson's blue	15 grains [1 grm.].
Alcohol (90 per cent.)	½ oz. [14·3 c.c.].

Dissolve and add as above.

For green :—

Naphthol green	½ drachm [2 grms.].
Glycerine	2 fluid drachms [7·2 c.c.].

Rub into a smooth paste, and add as above.

For brown :—

Bismarck brown ...	1 to 4 drachms [4 to 16 grms.].
Methylated spirit ...	3 fluid oz. [85 c.c.].
Glycerine	1 fluid oz. [3·6 c.c.].

Dissolve and add as above.

For yellow :

Acridine yellow	2 drachms [8 grms.].
Glycerine	1 fluid oz. [28·4 c.c.].

Dissolve and add as above.

Just before using, the melted gelatine mass is filtered through a double layer of filter paper. On an average 2 ozs. [56·8 c.c] of the gelatine mass are injected into each large artery, and at the end of the experiment the arteries are ligatured off after the brain has been put into cold water for a few minutes. It is then hardened in the following solution of formalin, to fix the colours—

Formaldehyde	1 pint [568 c.c.].
Potassic nitrate	2 ozs. [56 grms.].
Potassic acetate	1 oz. [28 grms.].
Water...	4 pints [2,272 c.c.].
Saturated aqueous solution of alum	10 fluid ozs. [284 c.c.],

for one month or longer, after which it is cut into sections in the horizontal, sagittal, or coronal planes.

The experiments consist (*a*) in injecting simultaneously three arteries, the anterior, middle, and posterior cerebrals,

with different colours after ligaturing the posterior communicating artery at one or both ends, and in some cases ligaturing also the anterior choroid artery; (*b*) in injecting four arteries, the three mentioned above and the anterior choroid; (*c*) in injecting five arteries, the posterior communicating along with the anterior, middle and posterior cerebrals and the anterior choroid, all on the same side; (*d*) in injecting either the basal or the cortical branches of the chief arteries alone.

I think that I may claim for this method that it would not be possible to inject the finest capillaries without using soluble colours; and owing to the communication in the cortex between the different areas, it would not be possible to keep the injections to their proper areas unless the same pressure was applied simultaneously to all the arteries. I found it possible to inject so small a body as the corpus subthalmicum with two colours, and to show that in some brains its two halves had a separate blood supply. All the colours were completely fixed by the formalin solution except the yellow, which washed out to some extent.

Basal Distribution.

I will now give the result of my injections, and will describe the various parts supplied by each of the arteries.

Taking the internal carotid first, and the supply of its branches in the order in which they are given off, the first artery is:—

The *posterior communicating* (fig. 11) which supplies the *optic chiasma*, the *tuber Cinereum* and the *regio subthalamica*, the part which is below the optic thalamus and is bounded ventrally by the posterior perforated space or the corpus mammillare and by the locus niger and pes pedunculi, medially by the third ventricle and the anterior pillar of the fornix, externo-laterally by the internal capsule and the reticular zone of the thalamus, and posteriorly by the nucleus ruber, while anteriorly it is continuous with the substantia innominata;

The *pes pedunculi* or crusta in its anterior one-third;

The *corpus subthalamicum* or Luys' body;

The part known as *Forel's field*;

The *anterior one-third* of the *posterior limb* of the *internal capsule*, below the level of the upper angle of the middle lenticular segment (figs. 4-6);

The *anterior external part* of the *external nucleus* of the *optic thalamus*, and sometimes the *anterior half* of the *internal nucleus*.

The next branch of the internal carotid is:—

The *anterior choroid artery* (fig. 12), which passes backwards and outwards to enter the anterior extremity of the descending cornu of the lateral ventricle, where the artery ends in the choroid plexus. On its way to the lateral ventricle it sends branches into the base of the brain to

The *optic tract*, which it supplies as far as its entry into the corpus geniculatum externum;

The *anterior one-third* of the *pes pedunculi* or crusta, sometimes, but less frequently than the posterior communicating artery; its *posterior two-thirds*, but less frequently than the posterior cerebral artery (*q.v.*).

Of the *internal capsule*, the *posterior* two-thirds of the *posterior limb*, as high as the superior angle of the second or middle lenticular segment (figs. 5 and 6);

Of the *lenticular nucleus*, the *first* or *internal segment*, the *third* or *external segment*, its posterior inferior part (sometimes);

The *nucleus amygdalæ* (fig. 11), within the hippocampal lobule;

The *anterior commissure*, in its extreme outer part;

The *choroid plexus*, the part in the descending cornu and the posterior cornu of the lateral ventricle;

The *choroid membrane*, the part in the descending cornu, anterior to the level of the coronal section through the posterior surface of the crus cerebri.

From these ventricular arteries of the descending cornu the roof of the lateral ventricle is probably supplied, and

The *retrolenticular fibres* (figs. 6 and 14), posterior to the internal capsule, including

The *optic radiations* at their origin (fig. 7);

The *tail* of the *caudate nucleus* or *surcingle*, in its inferior horizontal part.

The internal carotid divides into the middle and anterior cerebrals. I will take first the *anterior cerebral artery*, which supplies (fig. 10) :—

The *lamina terminalis vel cinerea*, forming the floor of the third ventricle ;

The *internal capsule*, anterior limb, the inferior half ;

The *head of the caudate nucleus*, inferior half ;

The *lenticular nucleus*, external or third segment, the anterior inferior part ;

The *lenticular nucleus*, second segment (fig. 9), the anterior inferior part, in about half the cases ;

The *septum lucidum ;*

The *anterior commissure*, median part ;

The *anterior pillar of the fornix*, upper part.

The other branch into which the internal carotid divides is :—

The *middle cerebral* (fig. 11), which passes outwards along the fissure of Sylvius giving basal branches at the anterior perforated spot, which supply

The *anterior commissure*, its outer part ;

The *lenticular nucleus*, the middle and the external segments ;

The *internal capsule*, the posterior limb above the horizontal level of the upper angle of the middle lenticular segment ;

The *internal capsule* (figs. 9 and 10), the anterior limb in its superior half (horizontally) ;

The *caudate nucleus*, the horizontal part posterior to its head ;

The *caudate nucleus*, head, superior half.

The two vertebral arteries join together to form the basilar, which divides into the two posterior cerebral arteries, each of which courses round outside the crus cerebri to the median surface of the temporal and occipital lobes, to end in the extremity of the calcarine fissure

The *posterior cerebral* artery supplies :—

The *corpus mammillare ;*

The *pes pedunculi* or crusta in its posterior two-thirds (fig. 12) ;

The *nucleus ruber;*

The *internal nucleus* of the *optic thalamus,* in its posterior half (fig. 14);

The *external nucleus* in its posterior half (fig. 14);

The *pulvinar* (fig. 14);

The *choroid arteries,* postero-lateral to the choroid plexus in the central lateral ventricle, and to the choroid membrane as far inferiorly in cornu descendens as the posterior surface of crus; postero-median to choroid plexus and membrane of third ventricle;

The *optic thalamus,* anterior superior part of the external nucleus (? from choroid membrane in central lateral ventricle); anterior nucleus (fig. 11) (? from choroid membrane in central lateral ventricle);

The *fornix,* body and crura posteriora; anterior pillar, inferior part;

The *corpora quadrigemina;*

The *corpus geniculatum internum;*

The *corpus geniculatum externum* (nineteen times in twenty-three cases).

Cortical Distribution.

According to my observations the most frequent distribution of the *anterior cerebral artery* along the median surface of the hemisphere is, that it extends posteriorly to about 1 in. anterior to the internal parieto-occipital fissure (fig. 16), and less frequently it extends as far back as this fissure, which is the extent given by Duret. Along the outer surface the area of the anterior cerebral artery extends posteriorly in most cases to half way along the parietal lobule (fig. 17), in place of the upper end of the fissure of Rolando as figured by Duret. Outwards and downwards on the external surface, anteriorly this area reaches to from $\frac{3}{4}$ to 1 in. from the median edge of the hemisphere and more posteriorly to the superior frontal sulcus (fig. 15); this is less extensive than according to Duret's description, which includes the middle frontal gyrus in the area of the anterior cerebral artery.

In the *middle cerebral area* the superior border of the anterior part is the same as that for the anterior cerebral as far posteriorly as the middle of the parietal lobule; posteriorly to this point the middle cerebral area either reaches the edge of the median surface of the hemisphere (fig. 16), or is bounded by the posterior cerebral area (fig 17), and this depends on whether the posterior cerebral area extends forwards anteriorly to the external parieto-occipital fissure (*vide infra*). The posterior limit of the middle cerebral artery on the outer surface, taken along a horizontal line, drawn from the fissura extrema of the calcarine fissure to the posterior angle of the parallel sulcus, is most frequently between the intraparietal sulcus at its posterior part, where it is continued into the anterior occipital sulcus, and the occipital pole. Nearly half the cases extend to the occipital pole, or to ½ in. anterior to it (fig. 15), in place of to just posteriorly to the upturned end of the parallel sulcus (Duret). The extension downwards of the middle cerebral area is to the middle of the inferior temporal convolution (fig. 18), or to the lower border of the middle or second temporal convolution, in place of to the middle of the second temporal convolution, as given by Duret.

The *posterior cerebral artery* supplies the whole of the median surface of the temporal lobe, and of the occipital lobe as far anteriorly as the internal parieto-occipital fissure, or to a point an inch anterior to this, except the most anterior part of the temporal lobe, which is supplied by the middle cerebral, and the anterior end of the gyrus uncinatus including the uncus, which is supplied by the anterior choroid artery, which anastomoses freely with the branches of the posterior cerebral artery to the rest of the uncinate gyrus (fig. 16). The posterior cerebral area extends round the occipital pole to the external surface for about ½ in., corresponding to the limit of the extension posteriorly of the middle cerebral area. More superiorly the posterior cerebral area extends across the median line on to the *outer surface*. In most cases it extends as far anteriorly as the external parieto-occipital fissure, which corresponds to Duret's description, or in some cases to half way along

the parietal lobule (fig. 17). The width of the area on the external surface is from $\frac{3}{4}$ to 1 in.

In relation to the extent to which the posterior cerebral artery reaches forwards on the external surface, is the area of extension of the anterior cerebral backwards and of the middle cerebral area towards the middle line. Out of fifty-one cases where it was observed, the middle cerebral area reached the middle line in thirty cases, and it failed to do so in twenty-one cases, so that the areas of the anterior and posterior cerebral arteries joined in these twenty-one cases, owing to the extension forwards of the posterior cerebral area.

The arterial supply to the corpus callosum (fig. 16) is from the cortical branches. The genu, rostrum and body are supplied by the anterior cerebral artery. The tapetum is supplied successively from before back by the anterior or middle cerebral, by the middle cerebral, and outside the cornu of the lateral ventricle by the posterior cerebral. The splenium and the forceps major by the posterior cerebral.

The supply of the centrum ovale (figs. 1-14) is from the medullary arteries of Duret, and the distribution of the arteries corresponds to that of the cortex external to it, so that the line of demarcation between the areas of distribution of any two arteries is quite hard and fast. I can corroborate by experiments Duret's statement that there is no connection between the cortical distribution and that of the basal arteries. I also agree with Duret that there is no anastomotic network in the pia mater, but that there is an anastomosis between the systems of the three chief arteries at the confines of their areas. This is shown by an experiment which I have made, and which consisted in ligaturing the third cortical branch of the middle cerebral artery, viz., that to the ascending parietal convolution, and then injecting the three chief arteries with separate colours with the same pressure. It was then found that the area supplied by the ligatured branch of the middle cerebral was injected from the anterior cerebral artery at its periphery rather than from the contiguous branches of its own artery, the middle cerebral. It is therefore difficult to understand why, when thrombosis occurs, the circulation in any branch is suffici-

ently interfered with to produce softening, unless very fine emboli block the capillaries of the cortex. I hold with Duret that the arteries which penetrate and supply the cortex are end arteries, and do not anastomose with their contiguous branches; and I have found that, if the pia mater be carefully removed, or a circular cut be made in it, the subjacent cortex is not injected by the vessels in the surrounding cortex.

Another point which is of interest is the supply of the middle and posterior cerebral arteries to the occipital white matter, and especially to the fibres of that part of the optic radiations which lie posteriorly to the part which, as we have seen (p. 409), is supplied by the anterior choroid artery. The relative share taken by these two arteries differs according to the distance that the middle cerebral area extends on the outer surface.

The most frequent distribution is the following:—

Taking a cross section of the optic radiations by making coronal sections of the occipital region, it is found that the superior three-quarter of the fibres, viz.: those fibres which supply the cortex of the cuneus and occupy the superior lip of the calcarine fissure, are supplied by the middle cerebral, with the exception of the posterior 1 in. (2·5 cm.), which is supplied by the posterior cerebral artery; while the whole of the inferior quarter of the radiations, which are considered to terminate in the cortex of the gyrus lingualis, are supplied by the posterior cerebral artery. The above distribution is found in those cases where the middle cerebral area extends to the intraparietal sulcus or to the posterior pole, but in those where the middle cerebral does not extend more posteriorly than the upturned posterior end of the parallel sulcus, all the fibres of the optic radiations in the occipital region are supplied by the posterior cerebral.

As the fasciculus longitudinalis inferior has of late years been regarded as the true course for optic impressions (Flechsig, Redlich [5], Archambault [1]), I have ascertained the arterial supply to these fibres in the occipital region, and I find that it corresponds to that given to the optic radiations, viz., that in most cases the superior part, as seen in coronal sections, is supplied by the middle cerebral, whereas the inferior part is supplied by the posterior cerebral artery.

References.

[1] Archambault. *Rev. Neurol.*, Nov. 30, 1905, p. 1,053.

[2] Duret. *Arch. de Physiol.*, 1874, t. 1.

[3] Heubner. *Centralb. f. d. Med. Wiss.*, 1872, Hft. 52; also "Die luetische Erkrankung der Hirnarterien," Leipzig, 1874.

[4] Kolisko. "Ueber die Beziehung der Arteria choroidea anterior zum hintern Schenkel der inneren Kapsel des Gehirnes," Wien, 1891.

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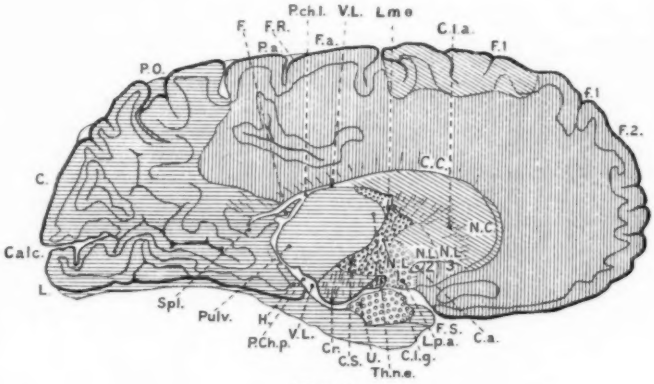

Fig. 1.

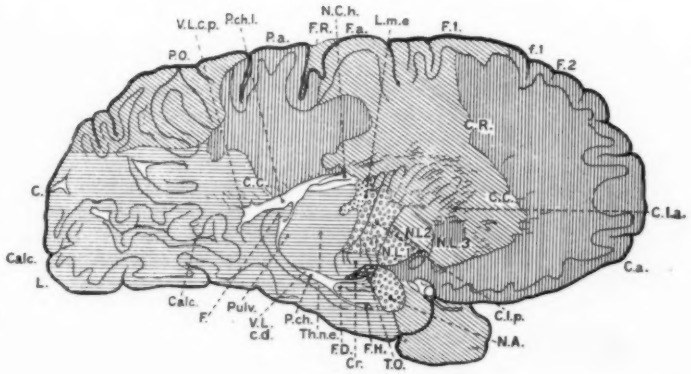

Fig. 2.

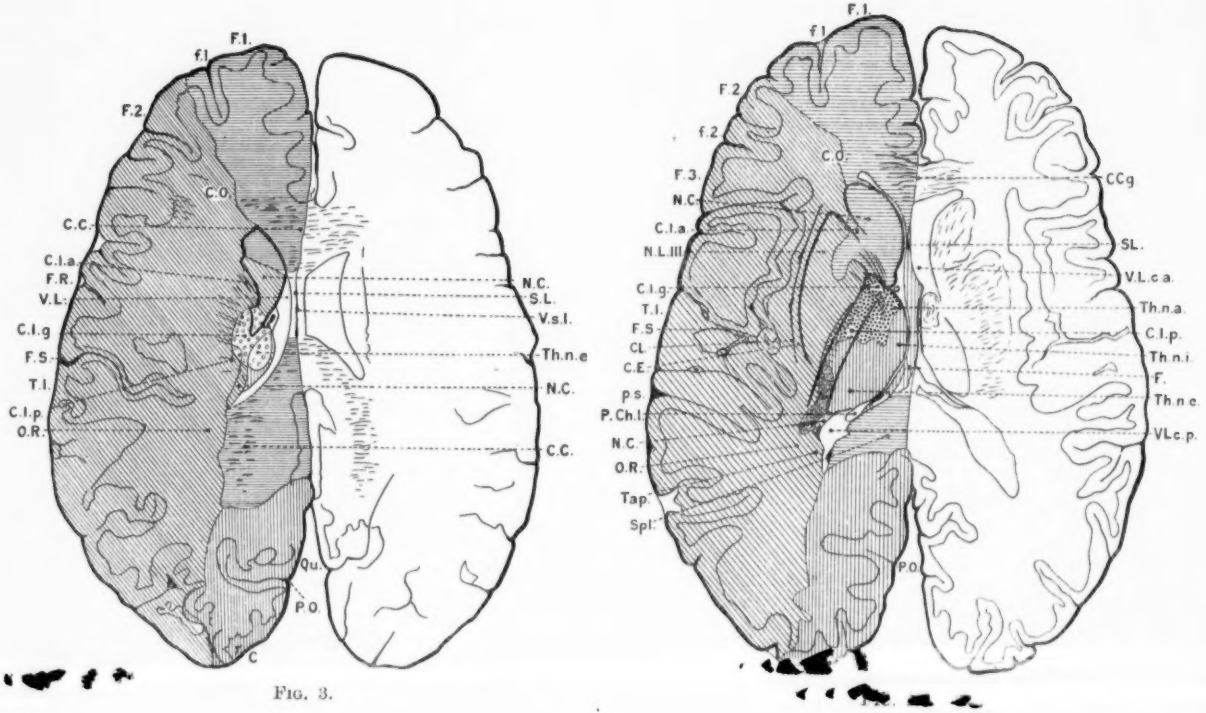
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FIG. 3.

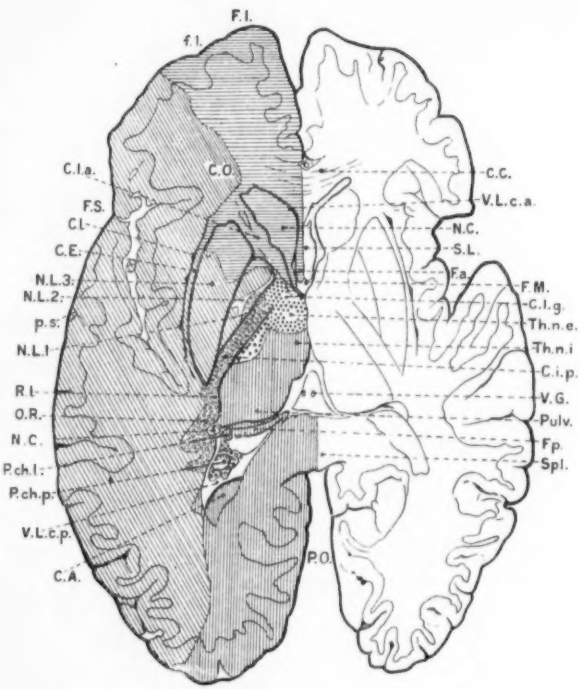
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FIG. 5.

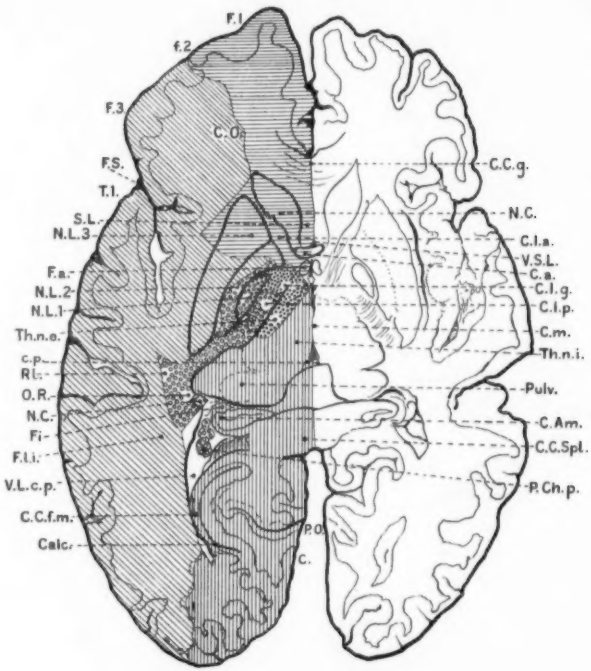
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FIG. 6.

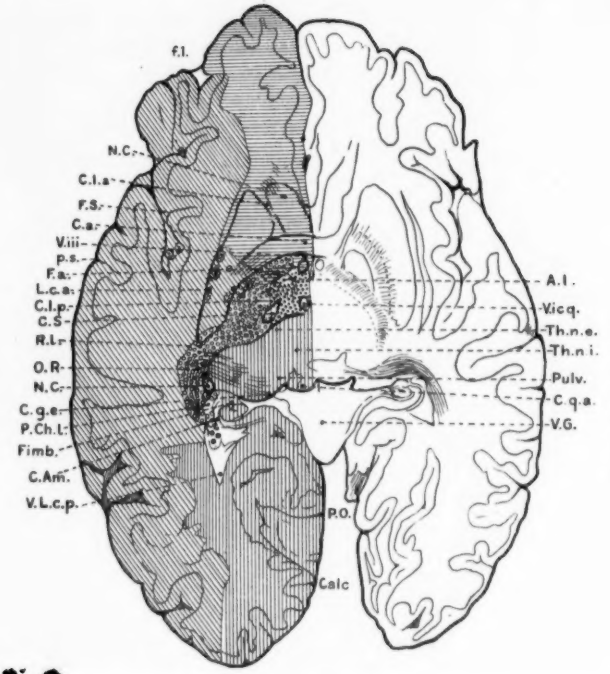
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FIG. 7.

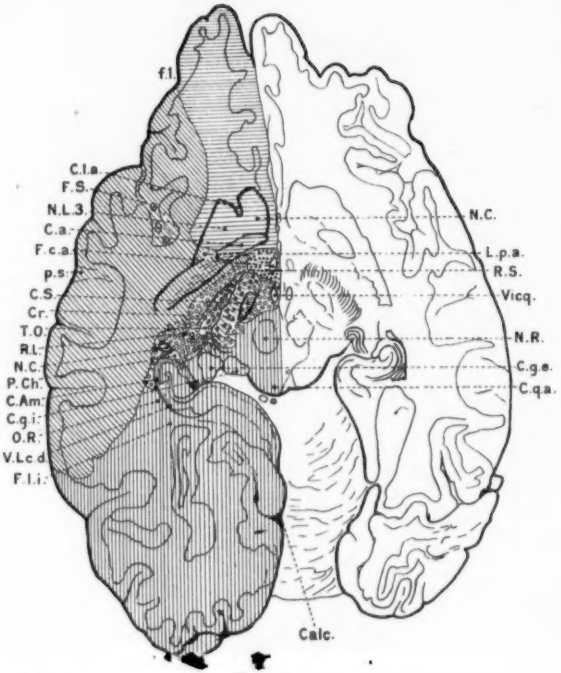
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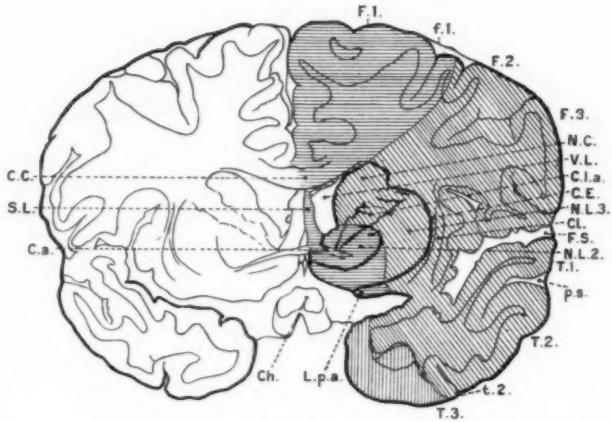
FIG. 8.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F.3.
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V.L.
C.l.a.
C.E.
N.L.3.
Cl.
F.S.
N.L.2.
T.1.
p.s.
T.2.
t.2.
T.3.
C.C.
S.L.
C.a.
Ch.
L.p.a.

FIG. 9.

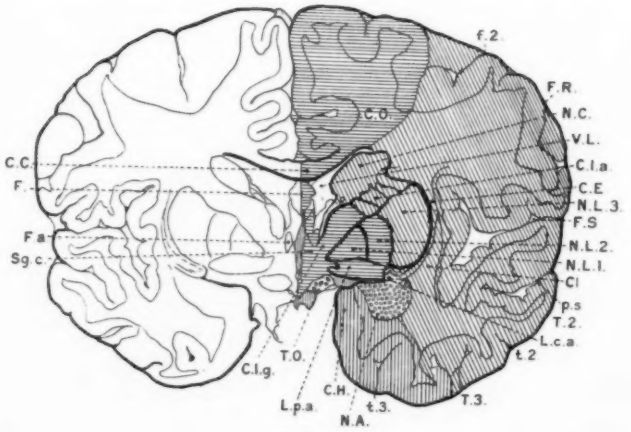
f.2.
F.R.
C.O.
N.C.
V.L.
C.l.a.
C.E.
N.L.3.
F.S.
N.L.2.
N.L.1.
Cl.
p.s.
T.2.
L.c.a.
t.2.
T.3.
t.3.
C.C.
F.
F.a.
Sg.c.
C.l.g.
T.O.
L.p.a.
C.H.
N.A.

FIG. 10.

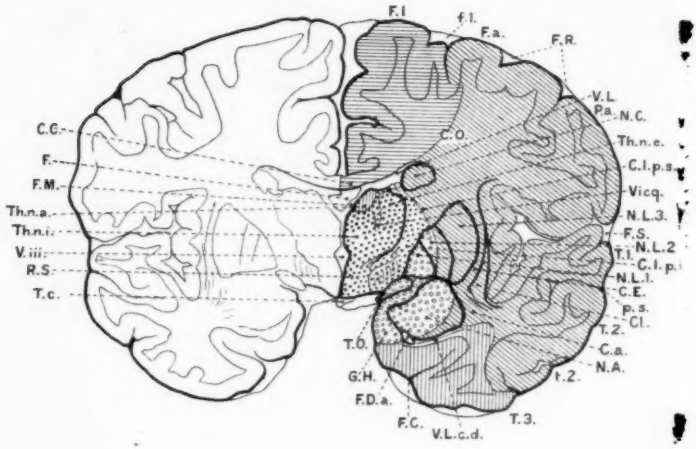
F.I
f.i.
F.a.
F.R.
V.L.
Pa.
N.C.
C.C.
C.O.
Th.n.e.
F.
C.i.p.s.
F.M.
Vicq.
Th.n.a.
N.L.3.
Th.n.i.
F.S.
V.iii.
T.1
N.L.2
R.S.
C.i.p.
T.c.
N.L.1.
C.E.
P.s.
Cl.
T.2.
T.O.
C.a.
G.H.
N.A.
t.2.
F.D.a.
T.3.
F.C.
V.L.c.d.

FIG. 11.

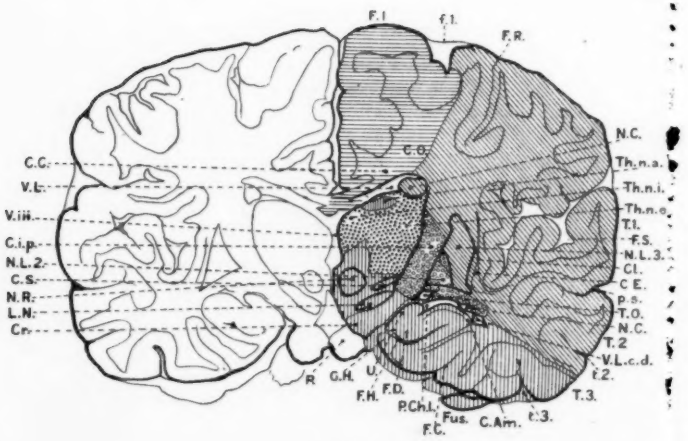
F.I
f.i.
F.R.
N.C.
C.O.
C.C.
Th.n.a.
V.L.
Th.n.i.
V.iii.
Th.n.e.
C.i.p.
T.1.
N.L.2.
F.S.
C.S.
N.L.3.
N.R.
Cl.
L.N.
C.E.
Cn.
P.s.
T.O.
N.C.
T.2
V.L.c.d.
t.2.
R
G.H.
U.
T.3.
F.H.
F.D.
P.Ch.l.
Fus.
C.Am.
t.3.
F.C.

FIG. 12.

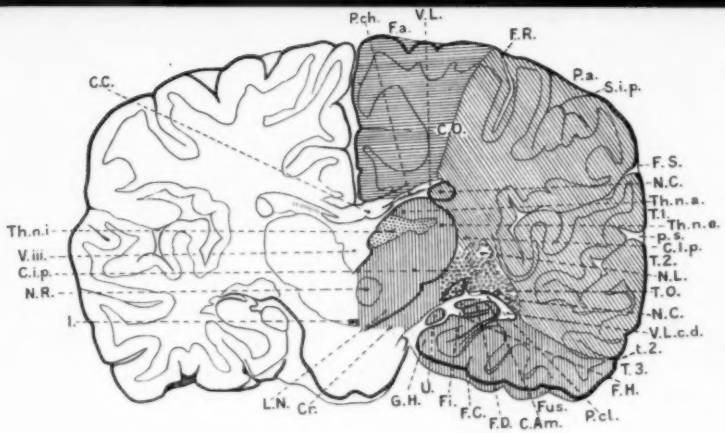

FIG. 13.

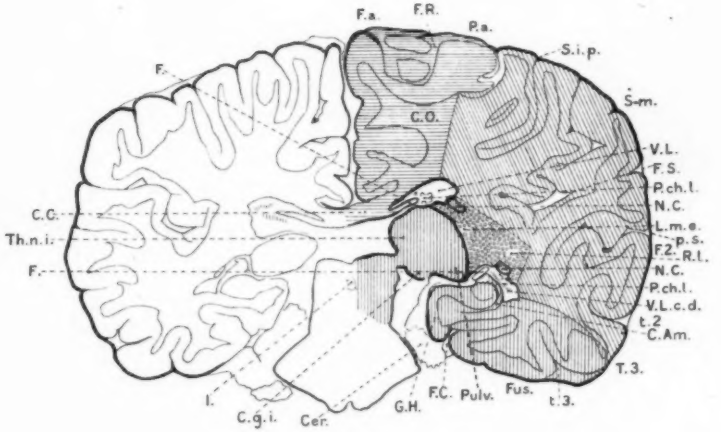

FIG. 14.

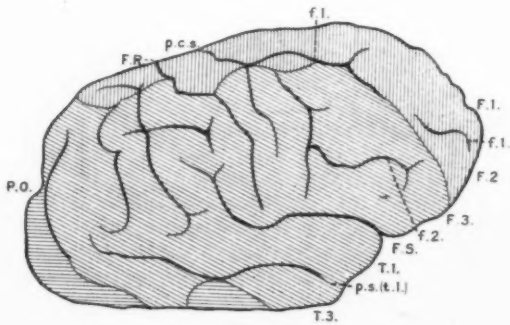

FIG. 15.

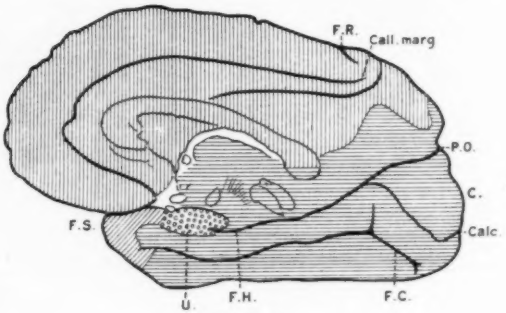

FIG. 16.

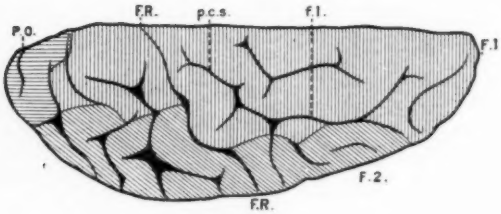

FIG. 17.

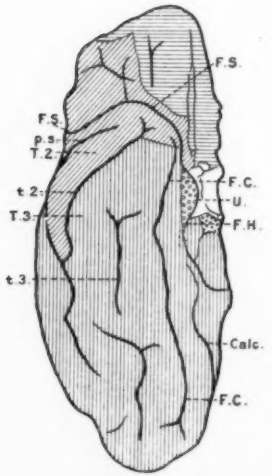

FIG. 18.

Description of Figures.

The figures are outlines of the photographs of the different injected sections and surfaces of the brain of man.

Figs. 1 and 2 are sagittal sections of the hemisphere made $\frac{3}{8}$ and $\frac{9}{16}$ in. respectively from the median surface.

Figs. 3 to 8 are horizontal sections of the brain. Fig. 3 is made through the most superior part of the optic thalamus; fig. 4 through the highest part of the lenticular nucleus; fig. 5 through the superior part of the internal segment of the lenticular nucleus; fig. 6 through the highest point of the anterior commissure; fig. 7 through the highest part of the corpora quadrigemina; and fig. 8 through the nucleus ruber.

Figs. 9 to 14 are coronal sections of the brain. Fig. 9 is made through the superior median part of the anterior commissure; fig. 10 is made just posteriorly to the optic chiasma; fig. 11 is through the tuber cinereum and the genu of the internal capsule; fig. 12 is through the most anterior part of the pons; fig. 13 is made just posteriorly to the lenticular nucleus; and fig. 14 is through the posterior part of the optic thalamus.

Figs. 15 to 18 are the external, median, superior and inferior surfaces respectively of the right human hemisphere.

In figs. 1 to 8 and 15 to 18 the area of the anterior cerebral artery is represented by lines drawn coronally across the long axis of the hemisphere. The area of the middle cerebral artery is represented by lines drawn diagonally. The area of the posterior cerebral artery is represented by lines drawn parallel to the long axis of the hemisphere.

The area of the anterior choroid artery is represented by small rings, and that of the posterior communicating by dotted shading.

In figs. 9 to 14 (coronal sections) the areas of the middle cerebral, anterior choroid and posterior communicating arteries are represented as above, but the area of the anterior cerebral artery is represented by horizonal lines, and that of the posterior cerebral artery by vertical lines.

Explanation of Figures.

(*The same letters apply to all the figures*).

A. l., ansa lenticularis.
C., cuneus.
C. A., calcar avis.
C. a., commissura anterior.
Calc., fissura calcarina.
Call. Marg., calloso-marginal fissure.
C. Am., cornu ammonis.
C. C., corpus callosum.
C. C. f. m., corpus callosum, forceps major.
C. C. g., corpus callosum, genu.
C. E., capsula externa.
Cer., cerebellum.
C. g. e., corpus geniculatum externum.
C. g. i., corpus geniculatum internum.
Ch., chiasma of optic nerves.
C. I.a., capsula interna, anterior segment.
C. I. g., capsula interna, genu.
C. I. p., capsula interna, posterior segment.
C. I. p. s., capsula interna, posterior segment, superior part.
C. I. p. i., capsula interna, posterior segment, inferior part.

Cl., claustrum.
C. m., commissura mollis.
C. O., centrum ovale.
C. p., commissura posterior.
C. q. a., corpus quadrigeminum anterius.
C. R., corona radiata.
Cr., crusta or pes pedunculi.
C. S., corpus subthalamicum (Luys' body).
F., fornix.
F. a., fornix., anterior pillar.
F. p., fornix, posterior pillar.
F. 1, gyrus frontalis superior.
F. 2, gyrus frontalis medius.
F. 3, gyrus frontalis inferior.
f. 1, superior frontal sulcus.
f. 2, inferior frontal sulcus.
F. a., gyrus frontalis ascendens.
F. C., fissura collateralis.
F. D., fascia dentata.
F. D. a., fascia dentata, anterior end.
F. H., fissura hippocampi.
Fi., fimbria.
F. l. i., fasciculus longitudinalis inferior.
F. M., fissure of Monro.
F. R., fissura Rolandi.
F. S., fissura Sylvii.
Fus., gyrus fusiformis.
G. H., gyrus hippocampi.
G. Hab., ganglion habenulæ of optic thalamus.
I., iter a tertio ad quartum ventriculum.
L., gyrus lingualis.
L. c. a., lenticulo-caudate arteries from middle cerebral arteries.
L. m. e., lamina medullaris externa of optic thalamus.
L. N., locus niger.
L. p. a., locus perforatus anticus.
N. A., nucleus amygdalæ.
N. C., nucleus caudatus.
N'. C'., nucleus caudatus, tail or surcingle.
N. C. c., nucleus caudatus, caput.
N. C. h., nucleus caudatus, horizontal part.
N. L. 1, nucleus lenticularis, internal segment.
N. L. 2, nucleus lenticularis, middle segment.
N. L. 3, nucleus lenticularis, external segment.
N. R., nucleus ruber.
O. R., optic radiations.
P., pons varolii.
P. Ch. l., plexus choroideus lateralis.
P. Ch. p., plexus choroideus posterior.
p. c. s., precentral sulcus.
P. O., fissura parieto-occipitalis.
p. s., parallel or first temporal sulcus.
Pulv., pulvinar thalami optici.
Qu., lobulus quadratus.
R. l., retro-lenticular fibres.
R. S., regio subthalamica.
S. g. c., substantia grisea centralis.
s. i. p., sulcus intra-parietalis.
S. L., septum lucidum.
S. M., gyrus supra-marginalis.
Spl., splenium corporis callosi.
T. 1, gyrus temporalis superior.
T. 2, gyrus temporalis medius.

T. 3, gyrus temporalis inferior.
t. 2, second temporal sulcus.
t. 3, third temporal sulcus.
Tap., tapetum corporis callosi.
T. c., tuber cinereum.
Th., thalamus opticus.
Th. n. a., thalamus opticus, nucleus anterior.
Th. n. e., thalamus opticus, nucleus externus.
Th. n. i., thalamus opticus, nucleus internus.
T. O., tractus opticus.
U., uncus.
U'., root of uncus where it is joined by fascia dentata.
V. iii., ventriculus tertius.
V. G., vena galeni.
Vicq., tract of Vicq d'Azyr.
V. L., ventriculus lateralis.
V. L. c. a., ventriculus lateralis, cornu anterius.
V. L. c. d., ventriculus lateralis, cornu descendens.
V. L. c. p., ventriculus lateralis, cornu posterius.
V. s. l., ventriculus septi lucidi.

THE STRUCTURE OF GREY MATTER.

BY JOHN TURNER, M.B.
Senior Assistant Medical Officer, Essex County Asylum.

Synopsis of Contents.

I.

Two classes of nerve-cells: (1) ganglionic or pale; (2) intercalary or dark. Similarly two kinds of neurofibrils: (1) smooth contoured, which proceed from the ganglion-cells; (2) beaded from the intercalary. This distinction is, however, only relative, as fibrils are continuous, therefore in places the one kind passes over into the other.

Cajal's primary and secondary neurofibrils.

Cajal's ammonia silver method displays the intercalary cells and (but very rarely) the beaded fibrils. This method, however, only exceptionally and imperfectly shows these structures.

II.

Description of the pericellular beaded fibrillar structure around ganglion-cells in the adult human subject, and the spinal-cord cells of the new-born rat.

Evidence adduced to show that the structure is a true network derived from the ultimate (fibrillar) arborisations of dendrites from the dark or intercalary cells.

III.

The evidence derived from anatomical, embryological, and pathological appearances favours continuity in the neurofibrils.

Scheme of nervous system on the lines of continuity, illustrating the relationship of the intercalary cells by means of the pericellular network to the ganglion-cells.

I.

By grey matter I refer to the grey matter of the central nervous system; that of the sympathetic and spinal ganglia does not react to my method, and therefore I am not in a position to make assertions as to its intimate structure. I believe that when these parts prove amenable to the method,

their structure will be found to be similar to that of grey matter elsewhere.

Golgi long ago drew a distinction between the various nerve-cells, placing in one class those in which the axis cylinder passes over into a nerve-fibre, and in the other those in which it is short and ramifies extensively; these latter are known as Golgi cells, or intercalated cells of von Monakow (*Schalt-zellen*). This is a distinction which rests entirely on anatomical features, and, moreover, its validity has often been questioned. In 1900 and 1901 I was able to demonstrate, by means of my pseudo-vital staining method, that in the cortex of the cerebrum and cerebellum of man there are two distinct classes of nerve-cells, distinguished not only by anatomical features but also by difference of chemical nature, in so much as the one class reacts to the stain in a different way to the other. Since then, in various communications, I have been able to show these two kinds of cell in the central grey matter, and in some of the nuclei of origin of the cranial nerves in man and rats, and recently I have met with successful results in the spinal cord of a two to three days old rat, showing here also the same distinction between the nerve-cells. One of these classes of cells, those the cytoplasm of which is barely coloured by the methylene blue, corresponds to the laminated cells of the cortex, the cells of Purkinje, the mitral cells of the olfactory bulb, to certain cells of the central nuclei, to the large cells of the cranial nuclei, and to the large cells of the spinal cord. These in future I shall term ganglion-cells. The other class, which stains very darkly by the methylene blue, is met with in all the layers of the cortex, in the molecular layer of the cerebellum and olfactory bulb, in the granular layer of the olfactory bulb (and probably, reasoning by analogy, in the granular layer of the cerebellum), scattered throughout the central nuclei, and among the large cells of the nuclei of the cranial nerves and of the spinal cord; in fact, so far as is known, in all grey matter. These I have termed dark or intercalary cells. The anatomical features which are common to this latter class are, that the dendrites spring abruptly from the cell-body, pursue a more or less

sinuous course for very long distances without any gradual tapering of the branch, which, however, is practically always moniliform or irregularly varicose; and that these dendrites either pass over ultimately into neurofibrils, on which are little clearly defined beads at relatively short distances from each other, or these fibrils may spring abruptly from the side of a comparatively stout branch (as rejetons). In the cerebral cortex their lack of orientation affords an additional means whereby they may be distinguished from the ganglion-cells.

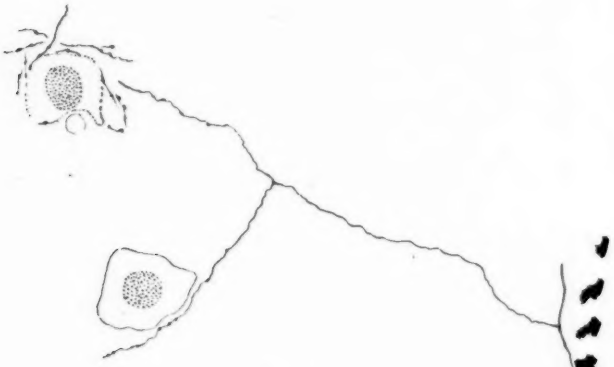

FIG. 1.

Human cornu ammonis (pseudo-vital method), showing the passage over of a smooth contoured neurofibril into a beaded fibril. Two ganglion-cells of the dentate fascia are outlined, and around one are portions of the pericellular beaded structure.

Furthermore, although my method does not show intracellular neurofibrils, it shows very distinctly a wealth of extracellular fibrils, which have tiny clear-cut beads arranged along them. Sometimes also sections show numbers of neurofibrils corresponding closely with those seen in sections treated by the methods of Bielschowsky, Bethe, Cajal, Donaggio, Joris and Lugaro; these are smooth in contour and devoid of the little beads, but after a long course they can be seen to pass over into beaded fibrils (see fig. 1).

In my opinion, these two kinds of fibrils come respectively from the two kinds of nerve-cells. The derivation of the beaded fibrils from the branches of the dark cells is capable of being easily verified by examination of sections treated by my method, and the derivation of the smooth contoured fibrils from the ganglion-cells is quite clearly shown by examination of sections treated by Cajal's method or those of the other investigators just mentioned. These are facts which do not admit of serious doubt; but whether the two kinds of fibrils are true varieties, or merely varieties produced by faulty technique, is a problem which yet remains to be definitely settled.

If, as I contend, neurofibrils are continuous, we should expect to meet with some which are in part beaded and in part without beads, and, as I have just stated, we do so.

From the beaded fibrils a pericellular investment is formed which stretches over each ganglion-cell, and in a later part of this paper I shall endeavour to establish that this structure is a true network, and not a mere collection of separate fibrils lying across the ganglion-cell.

As the "dark" cells possess anatomical characters which differentiate them from the ganglion-cells, other methods, providing the cells in question react to the stain, should show them, and to ascertain this I have recently examined a series of sections from both human and rats' brains, treated by Cajal's ammonia silver method, and have been able to identify some which evidently correspond to my "dark" cells, and in a few cases beaded neurofibrils proceeding from them. In the Cajal preparations, although it is possible to detect protoplasmic branches with the characters of those from dark cells, very often the cell-body itself is not visible, and is apparently unstained. I believe that this failure on the part of the cytoplasm to take up the colour applies also to the ordinary Nissl or hæmatoxylin preparations, and may be the reason why this class of cells has not been fully recognised.

As this paper is chiefly occupied with the subject of neurofibrils, it will be advisable to point out what constitutes, or is supposed to constitute, a neurofibril. Cajal

distinguishes within the cells two kinds, the primary and the secondary, the former are long, more or less thick (relatively), pass through the cell in a more or less direct manner, and are united to one another by finer, or secondary, neurofibrils to form a network. The primary fibrils show along their course darkly stained, fusiform swellings. Joris [8], in discussing the value of these two kinds, observes that many histologists (among whom he is one) will not allow that the primary fibril is a neurofibril, but is composed of several neurofibrils intimately joined together, and that the secondary fibrils appear between them every time a neurofibril separates itself from the bundle to unite itself with the neurofibrils of a neighbouring bundle. He believes the primary fibrils represent small bundles of neurofibrils which are agglutinated by the action of the silver nitrate, and he quotes an observation of Economo's which appears to bear this out; when a Cajal preparation is converted into a gold preparation it often happens that particles of reduced silver lodge between two fibrils, or mask several neighbouring fibrils, and these particles can be detached and removed by washing. The fibrils in one of these gold converted preparations are finer than in the original silver preparation, and the converted preparation closely resembles one treated by the method of Bethe. True (secondary) fibrils, according to him, preserve regularly the same calibre and volume, and do not show swellings; they follow a more or less undulating course, and do not make abrupt bends and angles; they divide and sometimes anastomose.

It might be argued that a fibril which divides is not a true (ultimate) fibril, but for all practical purposes I think Joris' conception of fibrils is sound, and that the primary fibrils of Cajal are really bundles of fibrils. If one compares sections treated by Cajal's method with sections treated by my method, it is seen that the delicacy of the extracellular fibrils in the latter is greater than in those of the former; and I believe, with very few exceptions, the extracellular fibrils in a Cajal preparation are bundles of two or more neurofibrils. It may possibly be due to this agglutination that beads are so rarely seen in preparations

by this method, but I am more inclined to attribute their absence to the supposition that Cajal's method selects the smooth contoured fibrils and fails to stain, except very occasionally and imperfectly, the beaded variety. One must not confound with the small rounded beads the slight fusiform varicosities which are frequently seen in Cajal preparations.

Joris believes that varicosities and beads are artefacts, the result of a defective technique. According to him, a perifibrillar substance which encloses the fibril retracts under the influence of divers reactions and accumulates from place to place, especially at the level of bifurcations.

The conditions under which the beads appear in sections stained by my method render such a conclusion, in my opinion, highly improbable. Thus it is only the neurofibrils which appertain to the dark cells which appear to show these beads, and they vary *constantly* in different regions, being largest and most clearly defined in the granule or stellate cell layers of the calcarine fissure region, and smallest and least defined in the molecular layer of the cerebellum. I am speaking now of the beads on the traject of the fibrils before they participate in the pericellular investment; in this structure their size is much more variable.

I have examined by Cajal's ammonia silver method the brains of two half-grown rats, one cut into sections sagittally, the other transversely; also sections from the prefrontal lobe, the ascending frontal, the calcarine area, cornu ammonis, thalamus and cerebellum in the human subject.

In the olfactory bulb of the rat, the dendrites of the mitral cells often show a moniliform contour, the only instance I have met with among ganglion-cells. Sometimes the swellings appear nearly empty, but usually fibrils can be traced across them. The neurofibrils in the cell-body and branches have an undulating course and are sometimes crinkled; they do not show lateral connecting fibrils, but in one or two instances I have seen a fusiform varicosity. The axons of the superficially lying mitral cells pass straight down through the molecular layer. At a short distance

from the cell-body they thin down considerably, and again a little further along become stouter and more darkly stained. They sometimes show slight fusiform varicosities (not beads) and sudden deviations from the line of traject and equally sharp returns, giving them a somewhat cork-screw-like appearance. These axons form a conspicuous feature in the molecular layer. I have seen no trace of a basket-work arrangement around the bodies of the mitral cells, such as is so clearly shown about the cells of Purkinje when treated by this method, neither have I been able to detect any of the small multi-branched cells in the molecular layer which my method displays, but I have seen darkly-stained, stout, sinuous branches containing fibrils, and sinuous fibrils on which are small varicosities, both of which, I believe, represent the branchings of dark cells. The small varicosities just mentioned are very like the beads seen on the fibrils springing from the dendrites of these cells, as shown by my method. The only other features noted in the molecular layer were fine darkly-stained fibres, some no thicker than a primary fibril, on which were very prominent oval, sausage-shaped, and round varicosities. A fibril could generally be seen passing longitudinally through these otherwise empty swellings. They closely resemble the apical branch of the granule-cells as seen when stained by my method, and there can be very little doubt but that this is what they are, although the bodies of these little cells are not stained by the Cajal method. They can be traced into the glomeruli and are then lost to view.

Although many smooth contoured fibrils occupy the granular region, I have not been able to detect any of the extremely fine, dichotomously dividing beaded fibrils which my method displays, and which form the axons of the granule-cells.

In the stratum oriens of the cornu ammonis of the rat, dark or intercalary cells are seen, giving off, as they do in preparations by my method, long, relatively stout, varicose branches, which show, but not very clearly, fibrils within them. In the stratum pyramidalum the ganglion (pyramidal) cells are stained yellow, and have a stout, gently tapering

apical process, which latter sometimes shows fibrils. A well-marked plexus of fine fibres, stained black, courses among the cell-bodies. Most of these fibres are stouter than the beaded fibrils which my method reveals in this region. They are of a smooth contour, with corkscrew-like twists on their traject, such as characterise axons; and in the stratum radiatum, adjacent to the stratum pyramidalum, they run for the most part in a sagittal direction, and form a plexus much richer than that seen between the cell-bodies. Other cells with the anatomical characters of "dark" cells are also met with among the ganglion-cells of this layer, but only very rarely. These, although generally oval or triangular, are less regularly disposed, they stain a deeper yellow, and their dendrites spring abruptly from the cell-body, are slender, sinuous and with well-marked moniliform swellings. Neurofibrils can be detected in the dendrites and cell-body (fig. 2). In the stratum radiatum, numbers of distinctly varicose fibres can be seen, which undoubtedly correspond to the branches of "dark" cells, which form so prominent a feature in this region in sections stained by my method. They are much finer than the apices of the ganglion-cells, and stain more darkly (fig. 3, D). The varicosities, fairly closely set, appear like oval rings across which a fibril can generally be traced. In some of the stouter branches the swellings are larger and more elongated; they may measure $5 \times 2\text{-}5\ \mu$.

I have not been able to determine with any certainty intercalary cells in the dentate fascia or in the stratum granulosum. In fact, although I carefully examined a number of sections, except in the stratum oriens and stratum pyramidalum, I was not able to find unquestionable representatives of the "dark" cells. Naturally the absence of a distinctive colour renders their detection more difficult, but if they were stained, their anatomical features should permit them to be recognised when carefully searched for. I believe that the great majority are not stained, and that only here and there the body (or more often some of the main branches only) happens to be selected.

In the human cornu ammonis I have also, with one or

two exceptions, failed to detect representatives of the "dark" cells. The fibrillar structure of the ganglion-cells is very well shown; the fibrils run in a wavy course from apex to

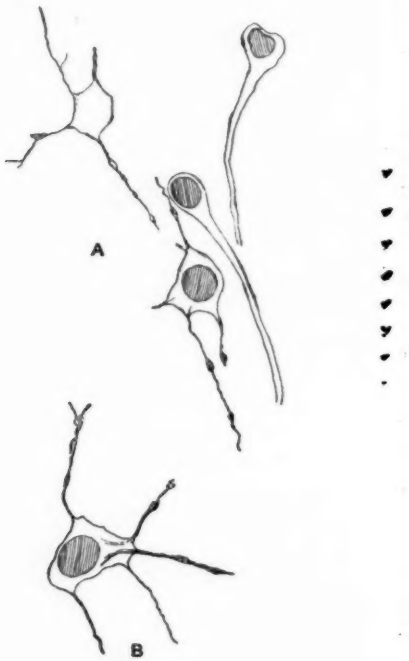

FIG. 2.

Rat's cornu ammonis (Cajal's ammonia silver method). A shows two cells with the characters of "dark" or intercalary cells, and two ganglion-cells to contrast with the two former. Notice the abrupt way in which the fine beaded branches arise from the cell-body, and that neurofibrils can be seen within the varicosities. In the lower of the two dark cells neurofibrils can be traced into the cell-body for a short distance.

B. Another cell with the characters of a dark cell, from same region. In this case the neurofibrils of two of the branches can be traced for some distance into the cytoplasm. Notice that the dark cells are larger than the ganglion-cells.

dendrites, are not varicose, but appear to give off lateral connecting twigs (secondary fibrils of Cajal), and in cells cut through in certain levels a perinuclear condensation ring

of fibrils is visible and a distinct fibrillar network, not only over the nucleus, but in more outstanding parts of the cytoplasm as well.

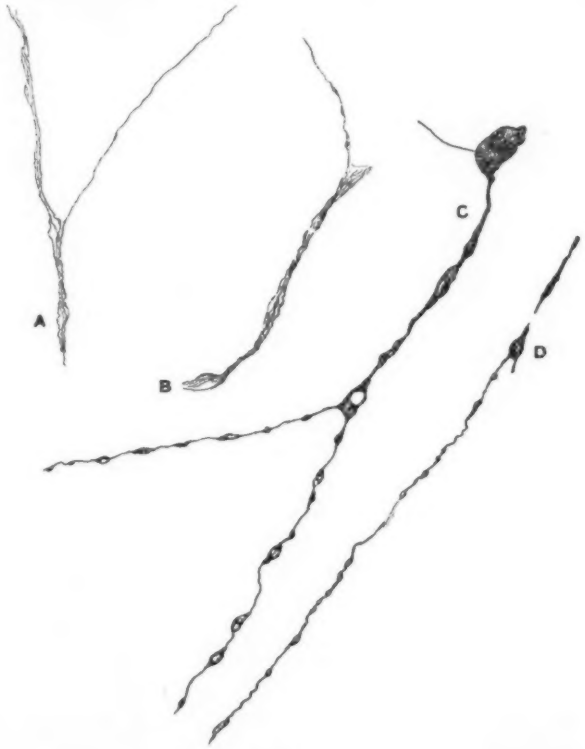

FIG. 3.

A, B (Cajal's method). Moniliform dendrites from cells (probably dark) in the thalamic region of the rat's brain. Notice the crinkled neurofibrils within the branches.

C (Pseudo-vital method). Moniliform dendrites of dark cell, from the same region as above.

D (Cajal's method). Moniliform dendrites in the stratum radiatum of the rat.

In the thalamic region of the rat, it is possible to pick out specimens which correspond to both classes. Those which correspond to the ganglion-cells are smaller, some-

what less deeply stained, and frequently bipolar, and they give off short straight branches which rapidly diminish in calibre. Those which correspond to the "dark" cells are more numerous, larger, more varied in shape, and give off very long dendrites, which do not rapidly diminish in calibre, and which are decidedly varicose.

Although the fibrils in the cell-body are not stained, they are very clearly shown in the varicose dendrites; they are wavy or crinkled. In the intercellular plexus of fibrils one can detect fine beaded twigs scarcely, if any, thicker than a primary fibril, lying among the smooth contoured fibrils.

In the human thalamus in transverse sections through the anterior tubercle the general appearance is very uniform; the cells are fairly regularly scattered, they are angular, square, or oval, and vary in size from 21 × 21 μ to 30 μ. The majority show fibrils which appear to be arranged in the form of a network throughout the cell-body. In sections which pass just above the level of the nucleus this network is exceptionally well shown, and there does not seem to be any special differentiation of the fibrils into a perinuclear net. In a very few, the more peripherally-situated fibrils appear to pass directly from dendrite to dendrite without visible anastomoses. In the stouter dendrites fibrils are visible, and some can be seen passing out into the intercellular plexus, and others can be traced into the bundles of axons which traverse this region. The dendrites do not show marked varicosities, so that it is difficult to say whether the cells from which they spring belong to the "pale" or the "dark" class; but the larger cells generally give off longer and more sinuous branches, and these cells probably represent the "dark" cells.

In sections of the rat's thalamic region stained by my method the varicose condition of the dendrites of the dark cells is a very marked feature. These varicosities vary in size, shape, and the distance apart from each other; they are usually oval with their long diameter in the course of the branch; they may consist of a slight and gradual swelling, or may quite sharply project out on both sides of

the fibre, and large ones are found situated a long distance from, while much smaller ones occur closer to, the cell-body. Many of them stain darkly all over, others have a dark rim with a pale or colourless centre. The general appearance of these latter is highly suggestive of their being originally independent cells which have fused together, end to end, as described by Fragnito [6] in the large ganglionic cells of the embryo cord. The very large size of some of these varicosities is opposed to the idea that they are a part of a parent cell-body. Thus in one case which I figure (fig. 3, c) a cell only 13 × 7 μ possessed a stout, moniliform dendrite which, after a course of 66 μ, broadened out into a somewhat triangular piece 10 × 4 μ, having in its centre a round pale area 3 μ in diameter ; from its free angles two fine branches ran, on which were numerous other large swellings with pale centres. The total bulk of these varicosities probably exceeded that of the cell-body. I believe these swellings represent the body, and the pale centre the nucleus, of originally independent cells which during the course of development have come to be fused together.

In the striate body of the rat stained by Cajal's method, whilst the majority of the cells consist of a well-defined nucleus, surrounded by a narrow clear zone, probably representing the portion of the cytoplasm destroyed by the treatment the tissue has been subjected to, a few are larger and denser, and their main branches contain clearly-defined fibrils which, entering the cell-body, spread out slightly and soon cease to be visible. No fibres with lateral twigs or rejetons were seen, although they are so prominent a feature in sections of this region stained by my method. It is not possible to say more than that probably the larger and denser cells referred to are representatives of dark cells. The fibrillar character of the matrix, although less dense than in the cortex, is well marked, and often individual fibrils can be traced into the strands of axon which traverse this region.

Whilst there is in sections of this region treated by my method no appearance of these rectilinearly coursing, smooth extracellular fibrils, the dark cells and the plexus

of beaded fibrils formed by the ultimate ramifications of their dendrites are beautifully shown. Detail of the finest character is revealed on the side of the dark cell system, but practically none on the side of the pale or ganglion-cell

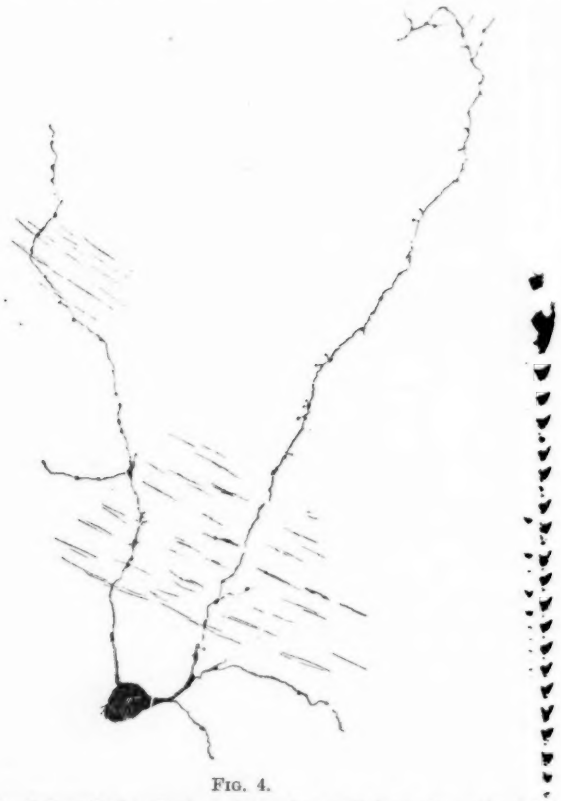

FIG. 4.

(Pseudo-vital method.) Dark or intercalary cell from the rat's striate body. The cell-body measures 10 × 8 μ. The longest branch is 182 μ. Notice the sinuous course of the branches and the beaded lateral offshoots or rejetons.

system. Owing to this selective action of the stain, the sinuous branches can be traced with the greatest ease. They are in the figure (fig. 4) represented in quite close

vicinity to their parent cell, no thicker than a primary fibril, and are beaded at irregular intervals, and throw off fine rejetons, which usually at a short distance terminate in a tiny knob, but which can sometimes be traced for long distances as beaded fibrils.

Cajal preparations of either sagittal or transverse sections of the rat's cortex are similar in appearance. There is a wealth of extracellular fibrils and fibres lying in all directions in all the cell layers, not appreciably richer in one region than in another, but as regards their local distribution they seem richest in those layers where the larger pyramidal cells occur. The molecular layer contains, except in the mesial aspect of the cerebrum, relatively speaking, few fibres, often distinctly beaded; this is a region, it should be noted, which is especially rich in beaded fibrils in sections stained by my method. The main trend of these fibrils and fibres is rectilinear, and, except in the outermost layer, I have not been able to detect any beaded fibrils in the intercellular plexus; but the number of fibrils is so large, and they are so densely packed, that it is quite possible I may have overlooked some beaded ones. In default of any colour differentiation, it would not be an easy task to detect them even if they were in places present.

A considerable number of the larger cells show fibrils arranged similarly to those in the cells of the cornu ammonis.

After a careful inspection of many sections, only one or two cells were observed that could with any degree of certainty be regarded as belonging to my dark-cell class.

In the human cortex I have examined many regions, but shall chiefly confine my remarks to a description of the fibril arrangement of the prefrontal lobe. In specifying this region it behoves one, in view of the amount of discussion which has arisen on its fibre distribution, to be very careful that the sections do correspond to this part and not to the adjoining frontal region. I cut my pieces about one quarter of an inch from the anterior extremity of the second frontal convolution, across the fronto-marginal fissure of Wernicke. The arrangement of the sulci in this region, however—and

it must be remembered that I am referring to the brains of those dying insane—is extremely variable, so that it is rare to find two which at all coincide. In these circumstances, whether my pieces in all cases included Wernicke's fissure I shall not insist on; at any rate I am certain that they were well within the area delimited by Campbell as prefrontal.

The following description of the fibrils is taken mainly from sections of this region from the brain of a low-grade epileptic imbecile. At the surface of the convolution the fibrils in the molecular layer are few in number, as this site is largely occupied by small glia cells, but in the parts which lie in the sulci the fibrils are thickly and evenly distributed throughout the entire molecular layer; they run chiefly in a direction parallel to the surface of the section, but here and there are long oblique fibrils, and others passing up from beneath at right angles to the surface. The majority are rectilinear in traject, and appear to be of quite uniform contour. In the second layer (small pyramidal) this arrangement alters, and they here form more of a plexus about the cells (of which only the nuclei are differentiated), lose their longitudinal course, are mixed with more perpendicular fibrils, and have, generally speaking, a more sinuous traject. In the third layer (of Campbell) the plexus grows more dense, and the individual fibres and fibrils again tend to run more rectilinearly. It must be kept in mind that the structures I am describing represent in many cases primary fibrils of Cajal, whilst others, although stouter and no doubt formed by the agglutination of more fibrils, are yet too fine to be measured with the eye-piece micrometer. The majority are, roughly speaking, not more than 0·3 to 0·5 μ thick. This plexus maintains much the same appearance in the lower levels, only here the strands of radiary fibres become more evident, although at their maximum these latter are insignificant features, as compared, for example, with those seen in the cortex of the calcarine region.

Only a very few of the cell-bodies show intracellular fibrils; rather more show them in their main branches. I have not been able to distinguish any cells corresponding to my dark cells, or, except in the molecular layer, any beaded fibrils.

In the cortex of the calcarine region, the intercellular fibrils, although abundant, are not more so than in the prefrontal. The greater development of the radiary bundles is the chief feature which distinguishes this region from the prefrontal as regards its fibrillar structure.

I wish to draw attention to the fact that the great wealth of extracellular beaded fibrils shown by my method in the cortex of the prefrontal lobe (*vide Brain*, 1906) is correlated with a wealth of smooth contoured fibrils, as shown by Cajal's method in sections of this region. The weight of evidence, as it seems to me, is against these being identical structures shown in a different form by different methods, as the course and arrangement of each kind is peculiar to itself. If my contention is correct that fibrils are continuous, one would necessarily expect to meet with this coincidence

Campbell [4] has called in question the accuracy of my deductions as to the fibril wealth of the prefrontal cortex, mainly because they do not coincide with the results which he finds in sections of this region stained by the Wolters-Kulschitzky process; but if he will examine sections prepared by Cajal's method he will find that all that part of the cortex which in his figures is depicted as devoid of fibres is in reality densely crowded with fine fibres and fibrils. The reason for this disparity is, I think, not far to seek. Campbell uses, for the detection of nerve-fibres, a method which does not directly stain these structures, but merely the myelin sheath which invests some only of the fibres. And as the fibrils and the great majority of the finer fibres met with in this region are devoid of a myelin sheath (or otherwise they would be revealed by the Wolters-Kulschitzky method), naturally he has failed to display them. On many occasions throughout his work he speaks of the presence or absence of nerve-fibres, whereas to be accurate he should have specified myelinated nerve-fibres.

In the cerebellum of the rat, only some of the bodies of Purkinje cells showed fibrils, in the dendrites, however, they were generally visible. It was sometimes not easy to determine whether the fibrils belonged to the dendrite, or were

climbing fibrils which followed the course of the dendrite like a creeper round a pole, and which apparently formed an extension of the basket system. The basket works and the system of stout parallel fibres from which they arise were well displayed, and these latter were seen to consist of bundles of neurofibrils. This is an important feature which my method does not show, and helps to explain the marked increase in calibre of these branches which occurs a short distance from the cell from which they appear to arise, and which, on the assumption of their fibrillar nature, was difficult to account for, if, as appeared in both Golgi's method and mine, they represented merely the axon of one cell. As a matter of fact, they represent axons containing fibrils common to many cells. Recruiting fibrils to these stout branches can often be seen, especially at the places where the latter make sharp bends.

In the inner third of the molecular layer there are large numbers of fine fibres and fibrils belonging chiefly to the basket system, but among them are fine beaded fibrils, the precise origin or destination of which I have not been able to determine. The middle third of this layer contains only a few fibres and fibrils, and the outer third is practically devoid of them; whereas in sections treated by my method these two last-mentioned sites contain large numbers of beaded fibrils.

I have not seen any small cortical cells, although nuclei which in all probability belong to them are stained.

In the human cerebellum the appearances are much the same. The cells of Purkinje and their branches show longitudinally running fibrils, but no clear appearance of a network in the body of the cell. Occasionally a small cortical cell is seen containing neurofibrils, but neither the cell or its fibrils are very clearly shown. As in the rat, the basket-works and parallel fibres are very distinct, and fibres coming from them are prolonged downwards to join in the formation of the plexus of fine fibres among the granules—the diffuse network of Golgi.

Although this investigation shows that cells having all the peculiarities of my dark cells are sometimes selected

by the Cajal method, they are, however, only imperfectly revealed. For whereas my method shows them to be very numerous in all grey matter, Cajal's method only picks out one here and there. Their remarkably long branches are rarely seen, and further, the beaded neurofibrils which I contend pass off from these branches are still more rarely shown.

In most of the regions, both in the rat's and human brain, where the fibrillar nature of the ganglion-cells was well shown, indications of anastomoses between fibrils were observed, in the form of a perinuclear net, or of a net extending throughout the cytoplasm.

II.

Probably every ganglion-cell—and I can now speak of those in the cord of the new-born rat—is invested by a fibrillar structure having the appearance of a network, and arising from beaded neurofibrils which pass off from the dendritic branches of the dark or intercalary cells.[1] This structure not only encloses the cell-body, but its dendrites as well, apparently throughout their extent; at all events I have been able to trace it around dendrites of the Betz cells for upwards of one hundred microns, at which length the branch left the plane of section (fig. 5).

The beads vary in size, shape, and construction: sometimes they do not measure more than 1 μ, sometimes 2 μ, or even more. They are usually round, but may be oval or triangular. Sometimes they appear as solid black bodies, at others as black rings (fig. 6, c). They are usually several microns apart on the parent fibril, but they may be nearly touching, or actually fusing together, in which case the investment seems partly to lose its fibrillar character, and to be formed of relatively stout fibres.

The complexity of the investment also varies very much. I am not now referring to its phylogenetic, but to individual

[1] The structure has been demonstrated by the vital method over the ganglion-cells of the thalamus in the rabbit by Turner and Hunter; but in my preparations of this region and the caudate nucleus it appears to be of a very rudimentary character, and consists of beaded fibrils which are not very manifestly denser over the cells than in the surrounding matrix.

variations, as seen in one section. In some cases only a few fibrils, with small, widely-separated beads, pass across the cell-body in various directions; in other cases the body of

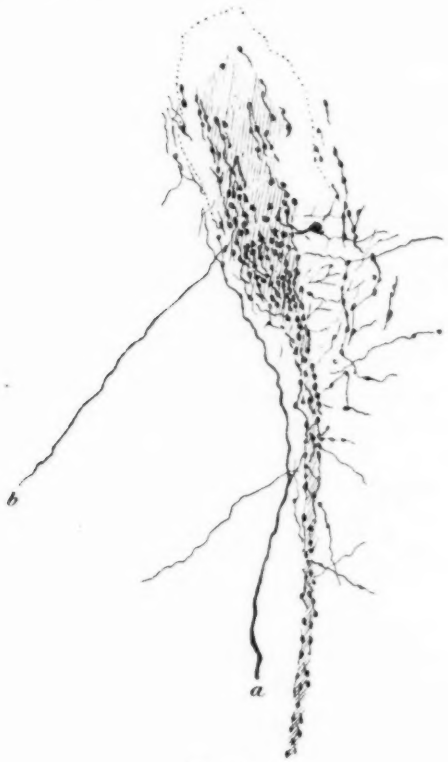

FIG. 5.

(Pseudo-vital method.) Part of a human Betz cell with a dendrite which can be followed for 180 μ before it leaves the plane of section, and which is encased all the way by the beaded fibrillar investment. Numerous fibrils pass to the structure from all parts of the surrounding matrix. *a* is probably a dendrite from a neighbouring dark cell; *b* probably a fine axon or collateral from a ganglion-cell. Only a portion of the dendrite is drawn.

the cell is nearly hidden by the densely-clustered beads over it (fig. 6 A). Quite possibly some of these differences may be due to pathological changes, for all my human material

has been taken from the brains of the insane, and I am inclined to think that in the case of the lower animals (cow, cat, rat, &c.) there has been more uniformity in the

FIG. 6.

(Pseudo-vital method.) Pericellular beaded fibrillar investments over human Betz cells (A, B, D), and over a solitary cell from the calcarine region (C). Details indicating the network character of the structure are shown in B and D. Notice the large size and frequent ring-like form of the beads in fig. C.

appearance of the structure. It is evidently very resistant to many morbid changes, and was well marked in some cases of senile dementia and advanced general paralysis.

The *Endfüsse* of Held, the buttons of Auerbach described by Cajal as seen with his new silver methods, and the buttons or "terminal knobs" described by van Gehuchten, are with great probability all identical with the beads shown by my method. van Gehuchten [17] states that the knobs by which the axis cylinders terminate round nerve-cells or their dendrites are always independent of one another, that they do not form a pericellular network, and are merely in contact with the cell. I believe, assuming that these *boutons* are the same as my beads, that the statement is incorrect in several respects. In the first place, although axis cylinders or collaterals do join to the beaded fibrils, or rather are continuous with them, this form of contact represents only a very small proportion to the number of cases where the beaded fibrils can be traced from the dendrites of the dark cells. In the second place, my method clearly shows that the beads are *not* terminal, and, except where the plane of section cuts across the traject of a fibril, in *all* cases one bead is joined to another by a connecting thread.

From the description given of the beads over the ganglion-cells by the various observers mentioned, it would appear as though they were only, or chiefly, encountered at these places; but it is one of the aims of this paper to point out that they occur all along the traject of certain fibrils, namely, that system of fibrils which has its extracellular origin at the points where the individual fibrils leave the *dendrites* of the dark cells *en route* for the pericellular investment.

And, lastly, I believe that there is good evidence to show that the pericellular investment forms a genuine network.

There has been a tendency for some to assume that the neurosomes of Held are identical with the beads I describe. But they are quite distinct structures. This idea may, to some extent, have been due to the photo-micrographic representations of this structure which I have published, because these pictures so often fail to show the connecting fibrils. Neurosomes are in the main internal constituents of the nerve-cell, but without question my beads are external, and lie usually at some little distance from the cell-body.

Scott [13] believes that the beads I describe are caused by heaps of neurosomes, but here again he assumes that they are merely terminal structures.

According to Held's figures, neurosomes occur as minute granules, somewhat sparsely scattered throughout the axis cylinder, in the axis cylinder hillock, and throughout the cell-body. In the axis cylinder hillock they assume a radial arrangement, and they are extraordinarily numerous at the terminals of many axons (this is Held's term, not mine), and because neurosomes are much less numerous in the dendrites and cell-bodies we have, he states, a ready method of distinguishing the protoplasm of terminal axons on cells (his so-called zones of concrescence). He figures a cell from the lumbar region of the cord showing concretions of neurosomes on its branches and axon, which certainly bears a superficial likeness to my beaded investment (see fig. 6, A), but on closer examination it is difficult to understand how the two structures can be confused. His little concretions consist of manifestly separate particles, and bear no real likeness to the sharply-defined homogeneous beads or rings shown by my method; and, above all, there is no sign of connecting fibrils between the neurosomes in Held's figure. My beads also do not lie in contact with the cell, but only in somewhat close approximation to it, so that focussing the investment flings the cell-body out of focus and *vice versâ*. Neurosomes are shown accompanying the axon of the cell, a position which in my preparations is devoid of the beaded investment.[1]

This structure is shown by Ehrlich's vital method; Cajal has evidently obtained it in methylene blue preparations, and a figure of his showing it around the cells of the posterior horn of the cord has been reproduced in many text-books as illustrating synapses, for example, in Starling's Physiology [15]. In Cajal's figure, collaterals are shown as if ending by extensive ramifications in a beaded structure

[1] The only exception I know of to this statement occurs in the basket-works around Purkinje cells. These basket-works, I believe, represent a modified but homologous stricture to that seen around the cerebral cells, and they form a densely matted tube which loosely encloses for a short distance the axons of the Purkinje cells.

around the bodies and main branches of the cells; but although collaterals undoubtedly are continuous with the structure, as I have shown in previous papers, I do not believe that this latter is merely a ramification of the collateral. The evidence previously given in this and other papers, showing that beads are a peculiarity of fibrils springing from the intercalary cells, strengthens this doubt. It is quite possible that the investment as he shows it is only partially revealed, owing to the method not selecting the intercalary cells, and that part of the structure which is directly continuous with fibrils from these cells.

It is a point of great consequence, in reference to the question of the general continuity of neurofibrils, whether this beaded investment consists of a number of separate neurofibrils from different sources, merely lying across the cell-body and branches in different directions, and with each of the fibrils ending free—that is, having no direct attachment to its fellow fibrils—or whether the structure is a true network formed by the organic continuity of the different fibrils. If this latter alternative can be proved, we have practically a demonstration of the continuity of neurofibrils, for it is an easily verified fact that going to (or passing from) the structure on all sides are numerous fibrils which originate from different cells.

It is essential, on account of the delicate nature of the fibrils composing the investment, to examine them with oil-immersion lenses of high magnifying power; otherwise it is easy to confound a mere overlapping with a real junction of the fibrils; and only after very careful searching have I been able to collect instances which stand the test of the highest magnification at my service, and which clearly indicate the network character of the structure. Some of these (figs. 6 B and D, 7 C) I reproduce.

It is, of course, impossible to assert positively that in every case what is shown as a junction may not merely be overlapping of two fibrils, although in many cases one can make practically certain of direct union when, as frequently happens (see especially fig. 6 *b* and *d*), a fibril meeting another more or less at right angles shows no trace, after

varying adjustments of the focus, that it is prolonged on the far side of the other. Another very common appearance which also strengthens the idea of fusion, is where several

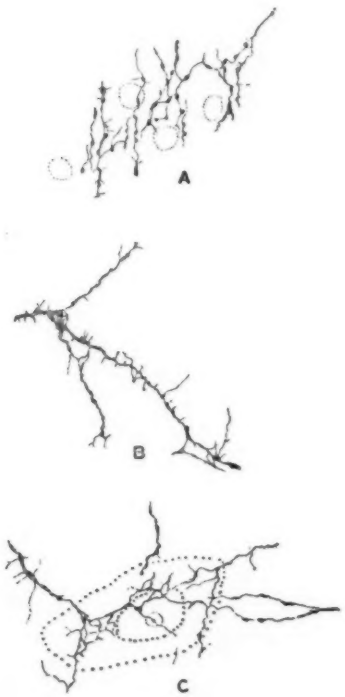

FIG. 7.

(Pseudo-vital method.) Spinal cord of new-born rat. A showing the network character of the beaded fibrils over the small pale nerve-cells (the nuclei only of which are shown) of the posterior root. B. Shaggy looking dendrites from dark cell, giving off numerous lateral offshoots or rejetons. C. The pericellular investment over a ganglion-cell, formed by the ultimate arborisations of branches from dark or intercalary cells, which freely anastomose with one another, and form a network.

beaded fibrils can be traced to a common fibril, joining their ends and forming the boundary of the investing structure over a cell-body and apex.

Further, W. Aldren Turner and W. Hunter [16], who studied this structure over the cells of rabbits, &c., as seen by the intra-vitam method, describe and figure it as a trellis or network stretching over the entire cell-body.

This appearance of the anastomosing of the beaded fibrils over the ganglion-cells was particularly well seen in

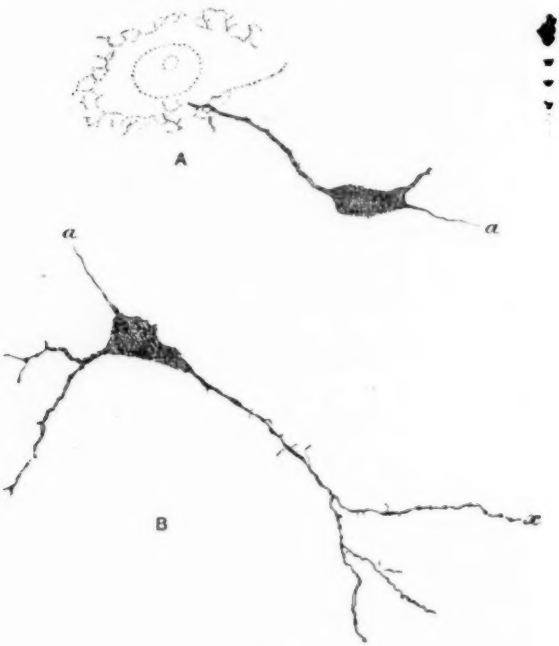

FIG. 8.

From same region as fig. 7, showing two dark or intercalary cells, with sinuous dendrites with rejetons, and very delicate axis cylinder (*a*). Notice the origin of the beaded fibril at *x* from a dendrite.

the case of a new-born rat, whose spinal cord reacted successfully to my method. In these young animals the fibrils are not so uniform in contour nor so distinctly beaded (fig. 7, c); they have rather an irregularly varicose aspect. The meshes of the net are more or less polygonal,

and the building up of the net by the union of fibrils coming from different sources is clearly seen. The fibrils are evidently in very close relationship to the Golgi nets, which in these specimens were somewhat vaguely depicted. The dark cells (fig. 8 A B) from which the pericellular fibrillar network arises occur in good numbers, and, although larger than those usually met with in the cortex, are small in comparison to the ganglion-cells of the fore horn (fig. 7 C, 8 A). Their nervous nature is beyond question, as their axons can frequently be seen. Their dendrites are long and sinuous, have a somewhat shaggy appearance near their origin, and sometimes consist for some distance of two parallel twigs, fusing here and there into a single branch. They present irregularly disposed, often long varicosities, which almost invariably give off at acute or right angles long, very fine, side twigs (rejetons), which can sometimes be traced on into beaded fibrils (fig. 7 B). The ultimate ramifications of the dendrites, and also these rejetons, form a somewhat coarse meshed network throughout the matrix of the grey, as depicted in fig. 7 A, where also are shown the nuclei of small ganglion-cells in the posterior horn, around which this network is disposed.

At a very early stage in my investigations (*Brain*, 1900). I drew attention to the derivation of the beaded fibrils forming the investment from the dendrites of the dark or intercalary cells. Since then I have been able to confirm this statement repeatedly, and in the *Journal of Mental Science*, 1903 and 1905, I gave photo-micrographs showing this mode of origin. It is not often, however, possible to get sections which, whilst showing this feature, are at the same time adapted for photography, and at the best such representations are not altogether satisfactory; for this reason I give two camera lucida drawings illustrating this mode of origin. They were both magnified about 800 or 900 diameters. Fig. 9 shows two dendrites from an intercalary cell in the ascending frontal cortex, giving off several beaded fibrils to the pericellular investments around three small ganglion-cells. Fig. 10, from a similar region in a different brain, shows an intercalary cell with an ascend-

ing axon. At one side of the cell a fine dendrite passes off, no stouter than a neurofibril which joins to the pericellular investment over the ganglion-cell. At a somewhat triangular-shaped bead it is apparently joined to a neurofibril coming from another source.

In commenting on Cajal's figure of beaded networks, I mentioned that I had also comparatively often seen a fine collateral or axon in continuity with the network, and also

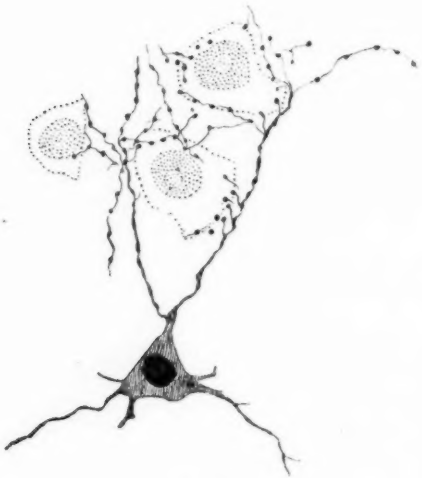

FIG. 9.

(Pseudo-vital method.) Motor cortex of man. Shows a small dark cell forming, by the ultimate arborisations of its dendrites, beaded fibrillar investments over three small ganglion-cells.

the direct continuity of a beaded with a smooth contoured fibril. These latter are usually not so darkly stained by my method, and at the place where the one variety passes over into the other there is frequently to be seen a short, dark, slightly swollen node, with rough or mossy edges. Sometimes, however, the point of junction seems to be a bead, for whilst on one side of the bead the fibril is darker, more sinuous and regularly beaded, on the other it is paler,

more rectilinear and free from beads. I have been able to trace a smoothly contoured neurofibril for over 200 μ without it showing any beads, and then there came a stretch on which, at distances varying between 2 and 4 μ, were beads, and at the same time the fibril became more sinuous. It is, however, generally at the networks that the one kind of neurofibril passes over into the other.

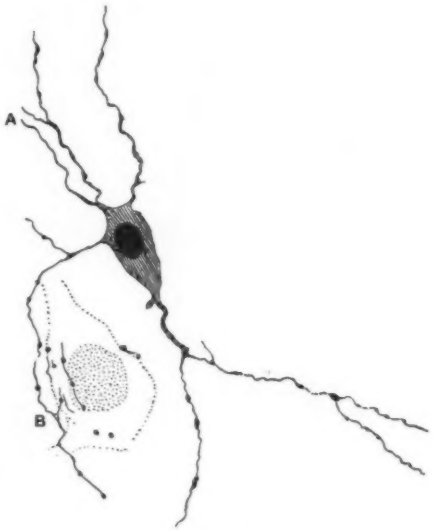

FIG. 10.

(Pseudo-vital method.) Motor cortex of man. Shows a beaded fibril coming from a dendrite of the dark cell, and passing to assist in the formation of the investment over the ganglion-cell. The appearance shown at B indicates the continuity of this fibril with one coming from another source. A. The axon of the dark cell.

NOTE.—All the figures were drawn with the aid of Zeiss' camera lucida. The two last viewed with a $\frac{1}{75}$ oil immersion, all the others with a $\frac{1}{12}$.

III.

In a previous paper (*Brain*, 1906) I briefly sketched a scheme of the central nervous system on the lines of continuity. I propose here to enter into, with greater detail, the construction of this scheme, in order to ascertain

whether it conforms to the laws of nervous conduction and degeneration. I have taken as my guide to the physiology of the nervous system Sherrington's [14] recently published work.

Apart from my own observations with the methylene blue and peroxide of hydrogen method, and which so far are largely unconfirmed,[1] I rest my belief in the continuity between nerve-cells on the following points :—

Certain embryological appearances, indicating the pluricellular origin of nerve-cells. This view receives support from the observations of most recent observers (A. Bethe [1], H. Joris [7], Fragnito [6], J. Cameron [2], R. Collin [5], according to whom the branches of nerve-cells, both dendrites and axons, are formed by the direct fusion of cells end to end, and at a later period the neurofibrils become differentiated within the individual cells, and in their turn fuse longitudinally one to another to form long continuous threads.

Appearances following degeneration. The idea that degenerative changes are restricted to the limits of particular neurones is probably erroneous. Many recent observers (von Monakow, Mœbius, Schäfer, Marinesco, Braunig, Warrington, &c.) find that this limitation has many exceptions, so that indeed some go so far as to state that a degeneration limited to one cell is the exception rather than the rule.

Continuity has been shown to exist in certain invertebrates which have been made the subject of special study (in leeches by Apáthy, in crustaceans by E. J. Allen, in rhizostoma, &c., by A. Bethe), which, from an evolutionary point of view, is strong presumptive evidence for its existence in vertebrates.

The presence of neurofibrils in cells. It is extremely

[1] This is true only to the extent that independent workers have not yet made use of the method. The sections of brain which show many of the appearances on which the scheme is founded—for example, the junction of the dendrites of the dark cells with the pericellular fibrillar investment—were shown at the meeting of the British Association in Belfast in 1902, and Professor Schäfer then stated that whilst he was unable to accept my interpretations, he could entirely corroborate my facts. Since then they have also been confirmed by other authorities.

difficult to frame a scheme of the nervous system on the lines of contiguity which is consonant with all the facts known as to the course and arrangement of the fibrils.

The following are, according to Sherrington, the differences between conduction in nerve-fibres and in reflex arcs :—

(1) Slower speed, as measured by the latent period between application of stimulus and appearance of end effect, the difference being greater for weak than for strong stimuli.

(2) Less close correspondence between the moment of cessation of stimulus and the moment of cessation of end effect.

(3) Less close correspondence between rhythm of stimulus and rhythm of end effect.

(4) Less close correspondence between the grading of intensity of the stimulus and the grading of intensity of the end effect.

(5) Considerable resistance to the passage of a single nerve impulse, but a resistance easily forced by a succession of impulses.

(6) Irreversibility of direction, instead of reversibility, as in nerve-trunks.

(7) Fatigability, in contrast with the comparative unfatigability of nerve-trunks.

(8) Much greater variability of the threshold value of stimulus than in nerve-trunks.

(9) Refractory-period (*bahnung*) inhibition, and shock, in degrees unknown for nerve-trunks.

(10) Much greater dependence on blood circulation; oxygen.

(11) Much greater susceptibility to various drugs; anæsthetics.

These differences, as Sherrington points out, appear referable to that part of the arc which lies in grey matter, and to account for them he relies, at any rate to a large extent, on hypothetical synapses, or intervening spaces where the terminals of one nerve-cell come into contiguity with the branches of another, and which space he likens to a mem-

brane not possessing reciprocal permeability. Such a device, granting that it exists and possesses the properties which he hypothecates, doubtless affords a reasonable explanation for the differences. But to those who believe in the continuity of the fibrils of the nervous arcs synapses do not exist, and for them some other mechanism must be found which will take the place of the synapses. Personally, I hold that in the pericellular fibrillar network over the ganglion-cells we have such an one, which is not only adequate, but possesses the advantage of being demonstrable.

It has now to be considered whether, granting certain hypothetical properties in nerve-cells, the scheme which is represented in fig. 11 suffices to account for the differences which exist between conduction in nerve-fibres and in reflex arcs. The full explanation of this figure will be given as I proceed to discuss the various functions of the central nervous system which it is intended to illustrate.

Whether we hold with continuity of neurofibrils or merely contiguity between the neurofibrils of different cells, there are certain laws governing the behaviour of nerve-currents which must be assumed, namely:

(*a*) That the intensity of a current in a neurofibril can be raised by an increase of the stimulus at the receptive end of the reflex arc, or by a blocking of the current at the effective end, or somewhere in the conductive path.

(*b*) That the intensity of a current can be lowered by a lessening of the receptive stimulus, or by drawing off the current somewhere in the conductive path or at the effective end.

(*c*) That the ganglion-cells represent stores of latent energy which can be communicated to the fibrils passing through the cells, hence currents issuing from ganglion-cells are of greater intensity than currents arriving at the cell either by its own intrinsic neuro-fibrils or by means of its pericellular investment.

(*d*) The special property which I would attribute to the pericellular network is that, under appropriate conditions, it is able to set free the latent energy in the (chromatoplasm of the) ganglion-cell, and thus reinforce the nerve-currents

passing through the fibrils of the cell; this property depending on the intensity of the nerve-current in the network.

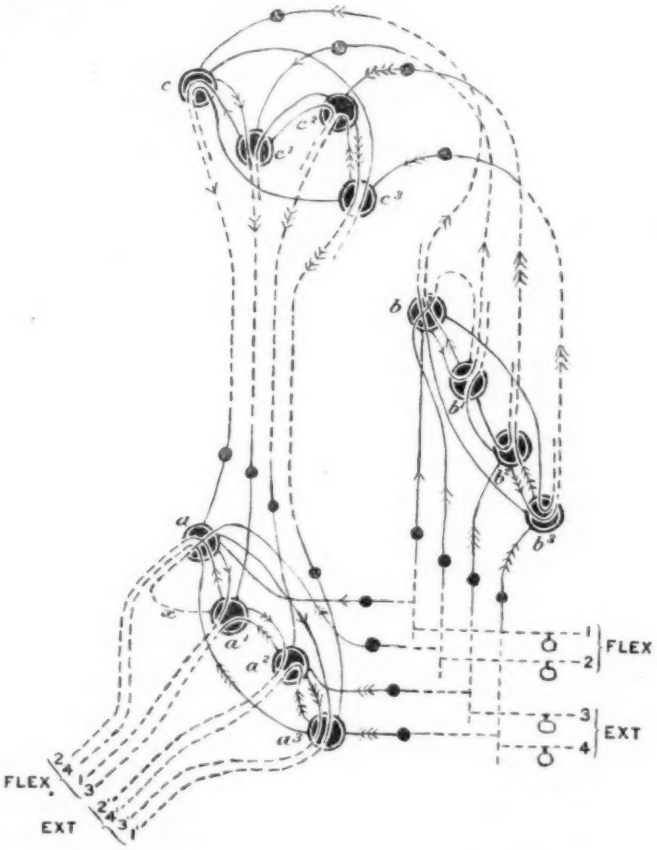

FIG. 11.

Scheme of nervous system on the lines of continuity of neurofibrils.

The first two of these postulates will probably be granted without question, and although there may be some who do not hold that ganglion-cells are stores of energy, there is

histological evidence in support of the view, in so much as this substance is used up during functional activity (Hodge, Mann, Vass, Donaldson). Robertson [12] on this point states, "the two special functional conditions to which definite structural changes may now be said to be proved to correspond are—(1) normal activity, and (2) exhaustion or fatigue. During normal activity the chromatoplasm is utilised by the cell and slowly diminishes in quantity. When the energy of the cell is exhausted the chromatoplasm is small in amount and appears diffusely granular, instead of forming local aggregations." The converse of this holds good, and during rest the chromatoplasm augments so that the cell assumes a densely stichochrome appearance (pyknomorphic cell).

Without definitely binding himself down to the chromatoplasm, Sherrington assumes that stores of energy are present in connection with, or adjacent to, ganglion-cells. "At each synapse a small quantity of energy freed in transmission acts as a releasing force to a fresh store of energy" (p. 155).

In the diagram (fig. 11) the intercalary cells are represented as round, black bodies, and their branches, both dendritic and axonal, as continuous lines; the ganglion-cells as circles, the ring representing the pericellular network, and the black masses within the chromatoplasm. Their dendrites are not shown; the discontinuous lines represent their axons.

As my method is silent concerning details of structure in the spinal ganglia, this portion of the scheme is merely provisional, but as regards the other parts I claim that I can demonstrate a coupling together of cells such as is shown; for example, the junction of the dendrites of the intercalary cells, and axons or collaterals of ganglion-cells (*x* in diagram), with the pericellular investment, are appearances which can frequently be seen in my sections, and which, from the point of view of continuity, do not appear to me capable of being incorporated into a scheme of the nervous system on these lines in any other way.

The scheme shows, for the sake of greater clearness,

merely a short (spinal) reflex arc, and a long arc including a subcortical and cortical centre. Granting the four just enumerated postulates, it will be found that it adequately accounts for the differences observed between conduction in nerve fibres and reflex arcs.

No. 1.—As regard speed of conduction, it is obvious that at each ganglion-cell station there will be delay, which will be in inverse proportion to the strength of the stimulus, and in direct proportion to the number of ganglion-cells included within the reflex arc. The delay will be caused at each ganglion-cell by the time required to raise the threshold of stimulus in the pericellular investment to such a strength as will enable it, by means of molecular disturbance of the chromatoplasm, to affect the nerve-currents in the fibrils passing through the cell in question.

Nos. 2, 3, 4, and 5 are equally obviously accounted for, and need not be considered in detail.

No. 6.—Irreversibility of direction, &c. This is a point which has had great stress laid upon it by the upholders of the neurone doctrine as favouring discontinuity. According to Professor Sherrington, irreversibility is due to the synapse, which he likens to a membrane not possessing reciprocal permeability; but it may be accounted for, according to the scheme, without invoking the aid of synapses.

So far as I can gather, this doctrine of irreversibility is based upon the Bell-Majendie law of the spinal nerve-roots, which states that stimulation of the central end of the motor nerve remains without obvious effect, and evokes no sign of reflex act or sensation; and also upon the fact, first ascertained, I believe, by Gotch and Horsley, that electrical variations will pass from the dorsal to the anterior root, but will not go in the reverse direction. If the anterior root $\frac{1.\ 2.\ 3.\ 4.}{1'.\ 2'.\ 3'.\ 4.'}$ is cut, it is clear that there can be no obvious reflex effects on stimulation of the central end. Motor effects are out of the question, except through possible paths coupling the cells of one segment with those of another, and even if nerve currents are able to pass in a reverse direction to that which they ordinarily take, these currents, instead of being intensified as they pass backwards,

will tend to become weaker, as the reinforcing influence of the pericellular investment on the chromatoplasm cannot take effect in the reverse direction. And further, the dispersion of the current through the different ganglion-cells on its course, and through the numerous intercalary cells and their branches, would render the attenuated remainder, which might ultimately reach the next segment or the posterior root in question, far too weak to excite motion or sensation.

Irreversibility, according to Bethe [1] and others, is not demonstrable in certain lowly forms of organisms—medusæ, &c., in which continuity in the nervous chain has been demonstrated, and although Sherrington cites this fact as supporting the existence of synapses it equally well accords with my scheme. The simpler the mechanism the more will reversibility show itself, probably even in vertebrates; as my scheme shows, assuming antidromic currents, there would be *some* amount of backward flow, but it would be so dispersed that its intensity at any one particular site would be too low to excite sensation or to register by the galvanometer. I would suggest that, with the rise in complexity of the pericellular network, the difficulty is augmented of passing currents in a reverse direction.

The fatigability in reflex arcs (No. 7), the (No. 8) much greater variability of the threshold value of stimulus, the (No. 10) much greater dependence on blood circulation, and (No. 11) greater susceptibility to drugs, are quite as readily and as adequately accounted for by my scheme as by one depending on synapses, and do not need further discussion.

The ninth clause, dealing with refractory period, inhibition, and shock, is the last one which remains to be considered. Inhibition and reciprocal innervation of antagonistic muscles will both be discussed together, as the latter implies inhibition.

The term "inhibition" is not altogether a fortunate one; it connotes a holding back, checking, hindrance, or repressing of energy. Now it is almost inconceivable that a stimulus, if effective, can hinder or repress energy—it must surely

liberate energy. But the energy set free is not necessarily displayed in muscular contraction; it may take the form of chemical changes, or it may be dispersed in various directions and so lose its demonstrable qualities. In the case of the vagus, one of the greatest stumbling-blocks in the paths of those who devise schemes of inhibition, there are facts which point to its inhibitory effects being rather a question of chemistry than nerve dynamics. Löwit [9] found that many soda salts in a proper concentration arrested the inhibitory action of the vagus, while potass salts restore the inhibitory action suspended by the soda salts. Compare with these observations the more recent experiments of J. Loeb [10] with contraction in medusæ. If the centre of a swimming bell (*i.e.*, a medusa in which the peripheral ring of nerve ganglia has been cut away) is put into a $\frac{5}{8}$ *n.* solution of sodium chloride it goes on beating rhythmically, but if a small quantity of calcium or potassium chloride, or both, is added it stops beating, showing, according to Loeb, that it is not the presence or absence of ganglion-cells which determines the spontaneous rhythmical contraction, but the presence or absence of certain ions.

In like manner it may be that when the vagus is stimulated there is a liberation of certain chemical substances in the muscle of the heart which prevent its contraction. Gaskell asserts that stimulation of the vagus ultimately improves the condition of the heart, as regards force, rate, or regularity, one or all of these, and groups it as an anabolic nerve, the temporary cessation of function indicating that constructive chemical changes are going on in the tissues.

Sherrington seems to be favourably inclined to this view of Gaskell's, for he "can imagine that a material continuously produced by a tissue, and yielding on decomposition the particular activity which is inhibited, may by an inhibition be checked in its decomposition, and accumulate, so that at the end of the period of inhibition the tissue contains more of the particular decomposable material than before. This molecular rearrangement would diminish activity for the time being, but lead to increased activity afterwards. There would ensue a rebound effect."

He takes exception to Macdougall's [11] theory of inhibition by drainage because it does not suggest to him a reason for this rebound effect, or superactivity as a consequence of inhibition; and also, a more serious objection in his eyes, because it seems to sever central nervous inhibition (*e.g.*, reciprocal innervation of antagonistic muscles) from other forms of inhibition met with peripherally.

But if, as has been contended, in the case of peripheral inhibition the effect is due to the nerve-current influencing some substance outside the bounds of the nervous system in the way of a chemical change, instead of directly in the way of a muscular contraction, and, as regards central inhibition, if it is due to a property inherent in the coupling together of ganglion and intercalary cells, whereby the passage of a nerve-current in *every* case is not only to augment energy in some paths but to withdraw it from others, I do not see where the force of either of his objections lies. In both cases the nervous processes are identical; in one case they give rise to a passive (central), in the other to an active (peripheral or extra-nervous), form of inhibition.

How does the scheme meet the requirements of reciprocal innervation? Sherrington has shown that the coördination in the taxis of skeletal musculature of vertebrates exerts its inhibition in the nerve-centres; that an increase of intensity, *e.g.*, of the plantar stimulus, does not make the spinal reflex action flow over from flexor to extensor muscles. The flexor reflex of the limb is unable to excite the extensor musculature, but, nevertheless, it has a coördinating influence on the latter. As regards flexion, *the skin fields whence the excitation (contraction) is elicitable, and that whence the inhibition is elicitable, are one and the same.* The same is found to apply also to cortical and subcortical stimulation. "The area of the capsular cross section at which the inhibition of the activity of, *e.g.*, the triceps can be evoked is separate from (not the same as) that area whence excitation evokes contraction of the triceps. On the other hand, the area of the section of the internal capsule, whence inhibition of the muscles is elicited, corresponds with the area whence contraction of its antagonistic muscles can be evoked."

Admitting for the present the validity of my postulates (*vide supra*), it is evident that *every* reflex act, according to my scheme, is inhibitory in nature. Let us follow the results produced by stimulation applied to the skin at 1, and for the moment confine ourselves to the short or spinal arc; it will set free a nerve-current along the branches of the spinal ganglion-cell, in the intercalary cell and its branches, and in the pericellular network of ganglion-cell a, passing thence down the neurofibril through the ganglion cell a^1 to the flexor muscle; a part of the current also will find its way to the extensor muscle along the neurofibril which runs through the ganglion-cell a^3. The paths of this current are indicated by a single arrow-head. This, however, is but a part of its action; the current flowing in the pericellular network of ganglion-cell a, will, it is assumed, by setting up molecular disturbance in the chromatoplasm, initiate currents of greater intensity in the neurofibrils passing through this cell, which, running down its axon (discontinuous lines), will cause contraction in the flexor muscle. This still does not exhaust the sequence of events which follows the original stimulus; the prepotent current in fibril 4 will, by drainage, lessen the intensity of the circulatory current in the pericellular network around ganglion-cell a^3 (it is presumed that currents of minimal intensity are always flowing along neurofibrils), and thus for the time being the threshold of stimulation of this cell will be raised, and there will be an inhibitory action on the tonal currents in the fibrils passing from this cell to the extensor muscle; that is to say, all the fibrils with the exception of the one which comes from the pericellular network of ganglion-cell a. In the scheme only two, or at most three, fibrils, are represented passing through the ganglion-cells, instead of, as is the case, very many, and therefore it might appear as though the greater intensity in the one fibril would neutralise the lessened intensity in the other; but, as a matter of fact, the large number of fibrils which are inhibited must greatly outweigh any effect which the single fibril might produce in exciting the extensor muscle to contract; and more especially would this be the case if the fibrils within cells anastomose with one another.

It will also be noticed that drainage will take place from the pericellular network around a^1; but the deficiency of intensity in the currents along the axon of a^1 which this drainage would cause will be more than counterbalanced by the intensity of the prepotent current the cell receives by means of the collateral of the axon of a (x in diagram), so that a discharge is set up in a^1; and, as a result, the flexor muscle receives a further incitement to contract. A similar chain of events will follow stimulation applied to the skin at 2; whereas stimulation applied to the skin at 3 and 4 will result in prepotent currents passing down the axons of ganglion-cells a^2 and a^3 to excite contraction in the extensor muscles, and drainage currents from a and a^1 inhibiting for the time being the action of these cells.

The scheme therefore provides that the site whence the excitation (contraction) is elicitable, and that whence the inhibition is elicitable, is one and the same; and exemplifies, as Sherrington maintains, that "The reflex excitation and the reflex inhibition are part and parcel of one excitation."

So far we have been dealing with short or spinal reflexes; when we turn to the long reflexes it will be seen that, although more complex (the currents at each interposed ganglion station tending more and more to diffuse themselves, and so to affect a greater number of cells), the general trend of events is similar; a stimulus setting free a nervous current in the uppermost spinal ganglion-cell, and flowing through the pericellular network of ganglion-cell b, will incite currents of prepotent intensity, part of which will find their way to the network round ganglion-cell c, and part to that round cell c^1, from thence to the flexor muscle *via* the axons of a and a^1.

The neurofibrils are shown as continuous from receptive to effective end, and their course is easily traced in the diagram by means of the arrow-heads drawn on them.

With regard to degenerative appearances in nerve-fibres as the result of experimental or other lesions, the scheme fully meets the requirements of pathological laws if we allow that the chromatoplasm of each cell presides over the nourishment of the fibrils which pass out of it along its

axon—a supposition which, as already stated, has much to be said in its favour.

I have not laid much stress on the intracellular arrangement of neurofibrils in ganglion-cells, chiefly because my method does not show them, and because whether they pass through cells independently, as Bethe contends, or form intracellular networks, as Donaggio and others contend, or exist in both forms, as Cajal, Joris, and others contend, does not seem to me to affect materially the working of my scheme. So far as I can arrive at a conclusion from my observation of Cajal preparations, I believe that most if not all ganglion-cells present a more or less well-marked perinuclear network (condensation); in some all the fibrils appear to anastomose, in others the more peripherally situated fibrils pass from axon to dendrite, or from dendrite to dendrite, without apparently forming any anastomoses.

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A FORM OF FAMILIAL DEGENERATION OF THE CEREBELLUM.

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SINCE the year 1893, when Marie [8] collated the cases which had been previously published by Fraser, Nonne, Sanger Brown, and Klippel and Durante, and formulated the conclusion that they all belonged to the same form of disease, for which he proposed the title *L'hérédo-ataxie cérébelleuse*, the subject of primary disease of the cerebellum has attracted considerable attention, and the title proposed by Marie has been adopted in many text-books as that of a definite clinical type of disease.

At the date of Marie's paper, in only two of the cases to which he referred had the nervous system been examined after death, in one of those reported by Nonne and in one member of Fraser's family; but since then the three cases recorded by Klippel and Durante and three members of the remarkable family which has been described by Sanger Brown have died, and the central nervous system of each has been submitted to a careful anatomical examination. In none of these cases, however, was any definite disease of the cerebellum found; in fact, disease of the cerebellum has been demonstrated in one only of the cases on which Marie's original paper was based, namely, in Fraser's.

A certain number of cases of primary disease of the cerebellum, some familial or hereditary, others sporadic, in which the nature of the disease has been determined by *post-mortem* examination, have, however, been recorded.

The cases described here are intended as a further contribution to the subject. For the clinical notes on the four cases and for the opportunity of examining the central nervous system of one of them after death I am indebted to the kindness of Dr. J. R. Lunn, under whose care the patients were at different periods in the St. Marylebone Infirmary. I wish to express my thanks here to Dr. Lunn for the further help he has given me and for the interest he has taken in this paper. Through his kindness I was also able to examine Case 4 some months before his death, and to get into communication with the surviving members of his family.

Neither parent of the family was afflicted with any form of nervous disease; the father died at 70 years of age, the mother, who was also a patient in the St. Marylebone Infirmary, at 89, with chronic rheumatism and bronchitis. There is no history of any relative, in either the ascendant or collateral lines, having been affected by any disease at all similar to that in the cases described in this paper.

There were eight children in the family; five of them were males, one of whom died in early life from heart disease, and of the remaining four, three, whose cases are to be recorded here, were afflicted with apparently the same form of disease. One of the three sisters also suffered with the same condition; the other two are still alive at the ages of 58 and 56, and are free from any of its symptoms. One of these whom I had the opportunity of examining kindly supplied me with further information on the rest of her family.

Case 1.—George S., the fourth member of the family, was admitted to the St. Marylebone Infirmary in 1886 at the age of 44. Prior to the onset of the symptoms to be described, he was healthy; he made good progress at school, and afterwards worked in a glass factory until he became incapacitated by his illness.

The onset of his symptoms was so slow and insidious that he could scarcely fix the date of their first appearance, but it was probably at about the age of 36 that he first noticed, or rather that it was pointed out to him, that he walked unsteadily or like a drunken man. It was only four years later, however, that this affection of gait gave him much trouble; from that time it increased slowly. At about 40 his arms also became unsteady and irregular in their movements; this symptom was also slowly progressive.

At the age of 42 his articulation was noticed to be peculiar and unnatural.

When he came under observation the patient was a man of small stature, with very little hair on his face, chest, or pubis, or in his axillæ. His penis and testicles were small and evidently poorly developed, and his tonsils were almost absent. His general appearance was described in the notes of 1886 as being "cretinoid." Sight and hearing were unaffected; his ocular movements were normal, there was no nystagmus; the pupils were equal in size and reacted well to light and on accommodation. His articulation was slow and drawling, his voice rather high pitched. He occasionally complained of giddiness. There was no loss of power in the upper extremities, though all their movements, but especially the finer ones, were poorly coördinated; "he cannot pick up a small object from the table quickly, and the more quickly he tries to do so the more he fumbles before he can pick it up. He spills water when attempting to drink out of a full glass." At that period he was able to walk without assistance, but only very unsteadily, reeling like a drunken man; he raised his feet unnecessarily and threw them quickly forwards. There was no tendency to Romberg's sign, that is, he did not become more unsteady in standing on closing his eyes. His knee-jerks were brisk, but no ankle-clonus was obtained. The sphincter functions were unaffected.

Two years later (1888) there was well-marked nystagmus on lateral movement of the eyes, and his other symptoms were further advanced. There was then also more fine tremor of the head and upper extremities. With the further progression of his symptoms he became bed-ridden and capable of doing but little for himself. He died some years later in the workhouse. In the interval he had been under Dr. Lunn's observation, but no notes were made on his condition.

It is interesting that this case, as well as his two brothers and his sister, when first seen, more than twenty years ago, was diagnosed as disseminated sclerosis; later, when Friedreich's ataxia was better recognised, they were regarded as atypical cases of this disease.

Case 2.—Robert S., the sixth child of the family, was admitted to St. Marylebone Infirmary in 1888 with chronic bronchitis. He was 40 years of age and had been, till then, employed as a labourer. The description of his condition was preceded by the statement that it resembled very much that of his brother and sister, who were then in the same institution. At about the age of 38 he began to stagger in walking, and somewhat later he

found his hands unsteady, especially in the finer movements, as in writing. He was also aware that his speech had changed.

His general appearance was apparently similar to that of his brother (Case 1), but there was more hair on his face, though but little on his body, and his external genitalia were also small and under-developed. No mention is made in the notes of any disturbance of vision or of hearing. Nystagmus was not then observed; his pupils were equal, and reacted to light and on accommodation. Articulation was slow and thick; this the patient himself attributed to his tongue being awkward, and getting twisted in his mouth. There was frequently some tremor of his head, especially when his eyes were closed; his tongue and arms were also tremulous. The movements of his upper limbs were strong, but definitely ataxic. His gait was unsteady and staggering, but the unsteadiness was not increased when he closed his eyes. His knee-jerks were brisk, but not definitely exaggerated.

He was discharged from the infirmary a few months later, but eventually returned. He was then scarcely able to walk without assistance, owing to the incoördination of his legs, and the movements of his arms were very ataxic. Articulation was so much altered that his speech was scarcely intelligible. He died in the infirmary a few years ago, but a *post-mortem* examination was not obtained.

Case 3.—Emma S. was the eldest sister of the other cases, and the second member of the family. The notes which were made on her case are not now obtainable, but in the clinical notes on each of her three brothers who were affected by the form of disease described it was mentioned that her symptoms were very similar, but further advanced than in them, and that she had in addition some of the symptoms of myxœdema. She was married, but had had no children. According to the information obtained from her sister, she had an attack of acute rheumatism when about 28 years of age, but had recovered and remained well till about 35; then her gait became unsteady, her hands awkward and irregular in their movements, and her speech altered. She was in the infirmary in 1886, when about 48 years of age, and was then apparently almost bed-ridden. She died some years later. Her condition was diagnosed as Friedreich's disease and myxœdema.

Case 4.—Benjamin S., the eldest member of the family, was admitted to the infirmary in 1890, at the age of 53. He had been previously in one of the London hospitals, where his disease was diagnosed as disseminated sclerosis. Till the onset of the illness

he had been healthy; he denied venereal infection, and had been a total abstainer from alcoholic drinks. When about 20 years of age he broke one of his legs, and never walked quite naturally afterwards, but it was only at about the age of 35 that his gait became unsteady; the difficulty in walking gradually increased from that time till he came under observation. At the age of 40 his hands were very unsteady and uncertain in their movements, and a short time later it was observed that his articulation was altering.

He was a small but well-built man, with very little hair on his scalp, and none on his face except his eyelashes; he had never found it necessary to shave. His sexual organs were, like those of his brothers, abnormally small and evidently under-developed; he had never married, and no mention is made in the notes of the state of his sexual instinct or power. His ocular movements were free, but lateral deviation of the eyes was interrupted by nystagmoid jerkings. The pupils were equal, and reacted to light and on accommodation. No mention was made of any affection of vision, but on ophthalmoscopic examination his optic discs were found normal. There was some tremor of his head when it was unsupported, and of his upper limbs. The latter, though they had lost none of their power, were ataxic and uncertain in movement. His gait was unsteady and reeling like that of a drunken man; Romberg's sign was not present. No disturbance of sensation was detected. The knee-jerks were brisk.

In 1905 he could scarcely walk alone; on one occasion he fell owing to an attack of vertigo. His disability had progressed, and his articulation had become slower and more jerky.

In 1906 (when seen by the author) he was bed-ridden, and unable to stand or walk without support. There was marked nystagmoid jerking of the eyes, especially on lateral movement, and his articulation was slow, jerky, and explosive. There was a considerable excess of contraction of the facial muscles when he spoke, similar to that so frequently seen in Friedreich's disease, and due, undoubtedly, to incoördination of the facial movements. The volitional movements of his limbs were very poorly coördinated, though not unduly weak; the ataxia was somewhat similar to that met with in Friedreich's disease. His tendon-reflexes were brisk, and there was no disturbance of the special senses or of sensation. He died in January, 1907, at the age of 70.

Autopsy.

The *post-mortem* examination was performed about thirty hours after death, in cold weather. The body was that of a

small, rather deformed man, with very little hair on the head, and none on the face or pubis. The penis and testicles were very small. His lower limbs were contractured in moderate flexion at the knees and hips. There was a general and more or less uniform kyphosis of the vertebral column in the upper thoracic region, and considerable scoliosis convex to the left of the thoracico-lumbar spine. The bones of the skull and vertebral column, as well as the dura mater and pia-arachnoid of both the cord and brain, were normal.

The spinal cord was of fair size, or at the most only very slightly smaller than normal, and no degeneration was visible in it on cross-section. On the other hand the medulla oblongata, pons and cerebellum, but more especially the latter, were very small; so small was the cerebellum that it lay almost completely within the natural concavity of the base of the brain. The brain-stem and cerebellum together weighed only 58 grammes, while the normal weight should lie between 155 and 180 grammes. When the pons and cerebellum were separated from the rest of the brain by transverse sections at the most frontal and the most caudal transverse fibres of the pons, their weight in relation to that of the whole brain was only as 1 to 16·3, while the normal proportion should lie between 1 to 6 and 1 to 7·5. By weight, therefore, the pons and cerebellum together were only from ·35 to ·45 of the normal; and if the cerebellum had been separated from the pons its proportional reduction in size would have been found considerably greater. The atrophy affected all portions of this organ, so that it remained approximately normal in shape; it appeared, in fact, a cerebellum in miniature (fig. 1).

When the pia-arachnoid was stripped off the cerebellum its folia appeared much atrophied, especially on the superior surface, and separated by unduly wide sulci (fig. 2). To the superficial view the superior vermis seemed to be the most wasted portion, while the tonsils, pyramid, and uvula appeared the least affected (fig. 3). The most striking feature of the cerebellum when viewed from the side was the unnatural exposure of the middle peduncle, owing to shrinkage of the folia on each side of the great horizontal fissure. The flocculus was very small, and the folia of the paraflocculus very wasted.

The reduction in size of all parts of the cerebellum seemed to be approximately the same. For comparison the cerebellum of a case was taken in which the cerebrum was equal in weight to that of this case, and the different lobes of the two were measured in exactly the same way. Then it was found that the proportion in

superficial extent of the various parts of the normal to the same parts of the abnormal cerebellum was approximately as 1 is to ·7. The difference was considerably greater in the superior vermis and flocculus, less in the tonsil and uvula.

In addition to its small size the cerebellum was unusually firm and hard, and to touch seemed undoubtedly sclerosed. Its superior and middle peduncles were approximately natural in size, but the corpora restiformia were certainly small.

The pons was found on measurement relatively less reduced in size than the cerebellum; its various diameters were approximately from ·75 to ·8 of the normal. The small size of the medulla oblongata was chiefly due to the smallness of the olivary protuberances and of the corpora restiformia. To the naked eye there was no apparent abnormality in the midbrain or the optic thalamus. The size and convolutional pattern of the forebrain were normal, except that the gyri of the prefrontal areas, on both the lateral and median surfaces of the hemispheres, were rather small and worm-like, suggesting a certain amount of general atrophy.

Microscopical Examination.

Cerebellum.—For the purpose of microscopical examination the cerebellum was divided by a medial sagittal section; the right half was separated from the brain-stem, and after hardening in alcohol was embedded in celloidin. Sagittal sections from it less than 1 mm. apart were stained by Nissl's method, and many of the intervening ones with hæmatoxylin and counterstained with picro-fuchsin or eosin. The left half, still attached to the pons, was mordanted in Müller's fluid, embedded in celloidin, and cut in the transverse plane of the pons; alternate sections were stained by the Weigert-Pal method, the others by hæmatoxylin or other nuclear dyes in order to demonstrate the condition of the cells and the ground tissue.

The changes which were found were identical and absolutely symmetrical in the two halves of the cerebellum. The only portions of it even approximately normal were the tonsils, the uvula, the nodule and part of the central lobe; the rest, both the vermis and the lateral lobes, was very intensely though not uniformly degenerated. In the molecular layer the change consisted in a reduction of its breadth, which in most places measured only from 100 to 150μ instead of 200 to 250μ, as in the normal (figs. 4-8); the majority of the cells which normally lie in it had disappeared, and it had undergone some sclerosis, evidenced

by a radial appearance due to a thickening of the so-called Bergmann's neuroglial fibrils; but this sclerosis was never great except in the deeper portion of the layer on the border of the intermediate zone in which the Purkinje cells should lie.

The affection of the granular layer was very distinct; it consisted in a considerable, though variable, loss of the granule cells, so that there was a marked rarefaction of the layer (figs. 4 to 8). Further, the nuclei of many of the cells which remained were angular or irregular in shape, or stained badly. In regions in which there was much atrophy the normal cluster arrangement of the granule cells was no longer recognisable. In consequence of this rarefaction of the granule layer, its outer border, instead of being, as it normally is, rather sharp and definite, was often ill-defined; in other folia, however, the more superficial granule cells were less affected than the deeper. Many of the acidophil structures of Denissenko could be seen in all parts of the granular layer; though relatively too numerous in the atrophied regions, there seemed to be an absolute numerical loss, but they were much less affected than the granule cells.

In addition to, and undoubtedly secondary to, this nerve-cell degeneration, there was a slight degree of neuroglial sclerosis throughout the whole granular layer wherever it was severely diseased.

The affection of the layer of Purkinje cells was still more prominent; in fact, in practically the whole cerebellar cortex, except in the tonsils, the uvula and the nodule, and in one or two small folia along the frontal border of the superior semilunar lobe, not a single Purkinje cell could be found, and even in these folia they were much reduced in number (figs. 4 to 8). Here and there masses of yellow pigment, with pale shrunken nuclei, were visible in the intermediate zone of the cortex; these possibly represented Purkinje cells, but they were not recognisable by their shape, and no processes could be seen. In many places a narrow belt of nuclei was distinctly visible in this layer, separated from the granular layer by a clear zone (fig. 8); they were distinguishable from those of the granule cells by their larger size and more irregular shape, and by the fact that, instead of staining intensely and almost homogeneously, they appeared paler in thionin and hæmatoxylin preparations, and one to three definite chromatin granules were often visible within them. They are undoubtedly the nuclei of proliferated neuroglia cells. Southard [11] has drawn attention to them in other forms of cortical atrophy of the cerebellum, and it was

probably these that Mott and Tredgold [9] described as forming an outer granular layer in a case of crossed atrophy of the cerebellum secondary to a cerebral lesion. Lannois and Paviot [6], who also observed them in cerebellar atrophies, regarded them as normal structures, which become more easily visible when the cells of the granular layer disappear. In many parts of the intermediate layer dense, compact strands of neuroglial fibrils were visible in even ordinary sections—as it was thought advisable to determine the exact extent of the disease in the cerebellum, no part of it was reserved for specific neuroglia staining—and wherever the Purkinje cells had disappeared there was, in addition, a more diffuse and general sclerosis of this layer.

The white substance of the folia was characterised by its greatly reduced size, and the van Giesen stain revealed a variable and often considerable amount of sclerosis within it (figs. 4 and 6). Masses of sclerosed glia often formed whorl-like clumps, and there was a tendency for these to surround the blood-vessels.

These degenerative changes were, on the whole, greater in the superior than in the inferior surface of the cerebellum, and they were perhaps most intense in the superior vermis, the tuber valvulæ, the flocculus, and in the pyramid; as a rule, the folia near the surface were more severely affected than those deep in the fissures. The tonsils were the most normal portions of the cerebellum; here the Purkinje cells were almost normal in number, especially in the ventro-medial folia, and there was but little atrophy of the granular and molecular layers, and less evidence of glial overgrowth than in any other part of the cortex. There were very few Purkinje cells in the uvula and nodule, and many of those which remained were atrophied; the atrophy of the granular and molecular layers was not so extreme as in many folia in which no Purkinje cells were found. The only other portion of the whole cerebellum in which any Purkinje cells were present was in one or two small folia along the frontal margin of the superior semilunar lobule, and in a few of the folia of the superior and inferior semilunar lobules near the bottom of the great horizontal fissure. In these folia there was less atrophy of the molecular and granular layers than elsewhere.

It may be said that the degeneration of the different layers of the cortex was proportional in degree, though it was not so everywhere; in some regions Purkinje cells persisted amid considerable atrophy of the granular and molecular layers, and in other

places where these were less affected no Purkinje cells could be seen.

The intrinsic cerebellar nuclei were about normal in size; the nucleus dentatus certainly appeared relatively too large for the size of the cerebellum, and all are well provided with cells, which did not appear diseased, though they contained a considerable amount of pigment, especially those of the dentate nuclei. This cannot, however, be regarded as a pathological condition, considering the advanced age at which the patient died, and the constant tendency of the dentate cells to accumulate such pigment. There was no evidence of sclerosis in the grey matter of these nuclei; the contrast between the degenerated cortex and the well-preserved intrinsic nuclei of the cerebellum was a striking feature in sections in which both parts were visible.

In addition to these changes in the nervous tissue, the walls of many of the blood-vessels of both the cortex and medulla were thickened, and their perivascular spaces were dilated and often contained a few infiltrating lymphocytes. This vascular disease seemed to bear no relation to the degeneration of the nervous elements; it was often most marked where this disease was slightest.

Sections stained by Weigert's method revealed a marked loss of myelinated fibres in the medulla of the folia of almost every part of the cerebellum (fig. 9). In some of the folia in which there was a large amount of atrophy of the cortex, practically no myelinated fibres remained, and in most of the others there was very great loss. It was chiefly the fibres of thicker calibre which had disappeared; the finer intrinsic (arcuate) fibres of the folia were less affected. As a rule, fewer fibres were found in the folia near the surface than in those deep in the sulci and in the neighbourhood of the central white matter of the hemispheres. In many folia practically no fibres stained in the granular layer, but in others the network of fibres in this layer was fairly preserved; the fibres of the Purkinje-cell layer were relatively less affected. The degeneration of the white matter was greatest in the culmen, pyramid, and in the anterior and posterior quadrate lobes, while the folia in which it was least affected were those of the uvula, nodule, the deeper portion of the central lobule, and to a less extent in the tonsil. The loss of fibres was thus roughly proportional to the degree of wasting of the cortex, but on the whole it was scarcely as much as might have been expected, considering the extreme degree of the cortical degeneration. There was also considerable atrophy of the white matter of the lateral lobes and

vermis; it was considerably greater in its dorsal and lateral portions, where the folia are attached to it, than centrally in the neighbourhood of the fourth ventricle, and in its ventro-lateral part, where the middle peduncles pass into it (fig. 9). The commissures of the vermis were much less affected than the rest of the white substance; large, well-stained, and apparently normal fibres could be traced from the dorsal commissure, dorsal to the dentate nuclei, into the region of the inner portions of the corpora restiformia; they correspond to the fibræ semicirculares of Stilling, and the fastigio-brachial fibres of Clarke and Horsley. Another bundle of fibres which was intact, or in which the atrophy was very slight, ran frontalwards from the frontal end of the dentate and roof nuclei in the ventro-median angle of the frontal third of the hemispheres; they apparently belonged to the *faisceau en crochet* and decussated in the anterior commissure.

In contrast to this marked atrophy of the white matter of the cerebellum the masses of fibres within the nuclei dentati, which further frontalwards form the superior peduncles, stained deeply and were apparently intact (fig. 9). There did not seem to be even the slightest degree of atrophy or degeneration of this peduncle. The fibres which surrounded the nucleus dentatus as a capsule and formed its fleece were very deficient in number, so that the fleece was extremely narrow, but the fibres which remained, stained more darkly than those of the rest of the white matter. The capsule of the nucleus tecti was also fairly well preserved. At the frontal end of the cerebellum there was a definite line of demarcation between the normally stained superior peduncles and the rest of the white matter, which was atrophied; in sections stained by picro-fuchsin this line was also recognisable, the tissue lateral to it being sclerosed, that medial to it, in which the superior peduncles ran, normal.

The middle cerebellar peduncles appeared intact, and all three layers of the transverse fibres of the pons which are continuous with them were well formed and apparently normal. No loss of myelinated fibres could be detected in the reticular substance of the tegmentum (fig. 9).

Spinal cord.—No tract degeneration or other abnormality was visible in the spinal cord in Weigert-Pal sections, except a slight relative pallor of the crossed pyramidal tracts in the lower thoracic and lumbar segments, but it was very slight, and nothing more than is frequently found in old persons with arterio-sclerosis. The dorsal and ventral roots and the dorsal root ganglia which were examined were normal. No degenera-

tive or atrophic changes were found in any portion of the grey matter which was stained by Nissl's method or by hæmatoxylin.

Helweg's tract, which could be followed upwards from the fifth cervical segment on the ventro-lateral margin of the cord, was constituted exactly as in the normal; there was no evident loss of fibres in it, though the pale area by which it is represented was slightly larger than normal. Nor could any sclerosis be detected within it. As it was traced upwards it grew larger and came to lie at the ventro-lateral margin of the median accessory olive, into the capsule of which part of it entered; the rest joined the ventro-lateral portion of the capsule of the inferior olive.

At this level the dorsal columns and their nuclei appeared normal, and well-stained internal arcuate fibres in normal number passed from the latter to the inter-olivary layer. The pyramids and the fibres of the dorsal and ventral spino-cerebellar tracts appeared unaffected as far as they could be followed through the brain-stem.

The inferior and accessory olives had undergone very striking alterations. Nerve-cells could be found only in the medial half of the ventral layer of the inferior olives, and even here they were atrophied, loaded with yellow pigment, and very deficient in number. In sections stained with thionin very little protoplasm was visible, and the nuclei were displaced and irregular in shape, apparently owing to the excess of pigment in the cells. The approximately normal portion diminished in relative size frontalwards; the contours of the olives in transverse section were traced through a drawing ocular at about the junction of their frontal and middle thirds, and on measurement it was found that the portion which contained cells was only about one-sixth of the whole. The portions from which all nerve-cells had disappeared was densely sclerosed and contained a great excess of neuroglia nuclei. This sclerosis was not limited to the lamina of grey matter, but also involved, though not in so great degree, the central white matter and the capsule of the olives. In the former it had led in places to so much shrinkage as to produce cavity formation. There was little or no sclerosis in the portions of the olives where the nerve-cells persisted. In the sclerosed portions of the olives large dense strands of neuroglial fibrils were found, similar to those which have been described in the Purkinje layer of the cerebellar cortex. A fair number of cells were present in the caudal end of the median accessory olive, but

elsewhere only a very few persisted in its ventral border. Not a single cell was found in any portion of the dorsal accessory olives. These were sclerosed in exactly the same way as the inferior olives.

The disease of the olives was quite as distinct in Weigert-Pal preparations. The normal folds of the grey lamella were recognisable only in the medial portion of the ventral layer (fig. 10); the rest was represented by an ill-defined area of pallor owing to the disappearance of the majority of the myelinated fibres which should form its capsule, as well as those of the white matter of the hilum.

Normally the capsule of the olives is formed of circumolivary fibres which are cut lengthwise in transverse cross-sections of the bulb, and which on the lateral and ventral surfaces are known as external ventral arcuate fibres, and by others which are cut transversely; the latter, which are most numerous on the lateral and dorsal surfaces, belong chiefly to the central tegmental tract. The whole capsule composed of these two sets of fibres was very much reduced in size. The deficiency was chiefly of the circumolivary fibres, and the capsule was consequently practically almost non-existent on the lateral and ventral surfaces of the olives. The transversely-cut fibres were less affected, and the central tegmental tracts, which could be traced frontalwards to the frontal part of the midbrain, were only very slightly reduced in size. The hila of the olives were intensely degenerated, and the majority of the fibres which pass transversely through the interolivary layers had disappeared; the small number of those which remained seemed to be in connection with the relatively normal ventral layer of the olives. The majority of the fibres which pass almost vertically through the dorsal layer of the olives from the region of the corpora restiformia were also absent; these are the fibræ olivo-cerebellares which spring from the cells of the olives and connect each olive with the opposite half of the cerebellum, the decussation taking place in the interolivary layer. The degeneration of the transverse fibres of the interolivary layers and of the hila of the olives was in large part due to degeneration of these. They were probably not completely absent, yet the majority of the fibres which intersected the dorsal laminæ of the olives were internal arcuate fibres from the dorsal column nuclei to the fillet layer. The myelinated capsules of the median and dorsal accessory olives were also much wasted.

There was abnormal pallor of the lateral portion of the

medulla oblongata, especially towards its frontal end, in the region between the olives and the corpora restiformia; this also was due to disappearance of the olivo-cerebellar fibres which normally pass transversely through this region on each side of the spinal trigeminus root. The abnormal smallness of the corpora restiformia must also be ascribed to the diminution in the number of these fibres. At the level where the corpora restiformia form compact bundles as they pass from the brain-stem into the cerebellum, the area of their cross-section seemed scarcely half the normal; it was kidney-shaped in outline, with the concavity pointing ventromedialwards. Owing to the shrinkage of the tissue it was difficult to trace the position of the degenerated fibres in the peduncle. The greatest loss of fibres seemed to be in its ventromedial portion, and a certain amount of sclerosis could be recognised in this region in van Giesen preparations. According to Thomas [12], however, the olivo-cerebellar fibres should occupy the lateral part of the peduncle, but van Gehuchten [4] believes that their position in the peduncle is not definitely known.

There was, however, a certain amount of degeneration and sclerosis in the outer portion of the peduncle in the medulla, though it was not so extensive or so striking as that in its ventro-medial part. In the cerebellum, too, it was the medial division of the peduncle that had most evidently suffered loss.

No pathological change was recognisable in any part of the grey matter of the medulla; the nuclei arciformes, the nuclei of the cranial nerves and the scattered cells of the substantia reticularis were unaffected. The nuclei laterales seemed intact and normal in size, though many of their cells were small and heavily pigmented. The cells of Deiter's and of Bechterew's nuclei were also normal in appearance and number. The pontine grey matter and the cells of the tegmentum of the pons were unaffected, and no evidence of sclerosis could be detected in the pons or midbrain.

In Weigert-Pal preparations the descending roots of the eighth and fifth nerves, the roots of the other cranial nerves, the dorsal longitudinal bundles, the fillet layer and the pyramidal tracts appeared normal. The transverse fibres of the pons did not appear reduced in number or atrophied, and all the other systems were quite normal.

In the midbrain the superior cerebellar peduncles appeared unaffected and perfectly developed; their fibres could be followed through their decussation and to their termination in the red

nuclei and the optic thalami. Both the grey and white matter of the latter, as well as the cells of the red nuclei, were normal. There was no degeneration in any portion of the crura. No evidence of degeneration was revealed by Weigert's method or by any of the other stains used in the internal capsule or in the basal ganglia.

Portions of different areas of the cerebral cortex were examined by both Nissl's and by Weigert's methods. By the former nothing abnormal was observed in any region, except perhaps a slight reduction in size of the Betz giant cells of the precentral area, and a considerable amount of pigment in the cells of all areas; this excess of pigment can scarcely be regarded as pathological in the brain of a man 70 years of age. The myelinated fibres of all regions were unaffected.

The optic nerves, which were also examined by Weigert's method, showed no sign of atrophy or degeneration.

The peripheral nerves which were examined were also normal, and the only change discovered in the muscles was a slight reduction in the diameter of their fibres, such as might be expected in the case of long disuse.

To summarise these facts, we have the history of a family with eight members, of whom four were afflicted with the same symptoms. In each they set in between 33 and 40 years of age and progressed slowly till death, which resulted from an intercurrent affection at an advanced age. The first and most prominent symptom was a reeling or staggering gait, "like that of a drunk person," evidently due to incoördination of the movements of the lower limbs or to defect in equilibration; the next symptom to appear in each case was irregularity in, or uncertainty of, the movements of the upper limbs; and later articulation was affected, it became hesitating or scanning and explosive. Tremor of the head and limbs was mentioned in the notes of all the cases, and was referred to as a prominent symptom by the sister who was interviewed. Nystagmus, or more probably irregular nystagmoid jerkings of the eyes on movement, was observed in the three cases of which accurate notes are available. There was no defect of vision or of hearing, and no motor paralysis or sensory disturbance was detected. The tendon-reflexes were unaffected

or slightly exaggerated, but ankle-clonus or other certain evidence of disease of the cortico-spinal tracts was not obtained in any of the cases. The sphincter functions were intact, and there was no obvious mental impairment.

The anatomical basis of these symptoms, which it may be assumed was the same in the first three cases as in Case 4, was a degenerative affection of the cerebellum. The disease was limited to this organ, as the inferior olives were the only other part of the nervous system in which pathological changes were found, and in these it will be shown that it was only secondary and of no significance in the production of the symptoms.

There can be but little doubt as to the nature of the pathological process in this case. Both its clinical features and its morbid anatomy point to the disease having been a primary and progressive degeneration of the nervous elements of the cortex of the cerebellum and of the fibres which spring from them. The most prominent change in the cortex was the disappearance of the Purkinje cells, which was complete in the greater part of the cerebellum, but it seems impossible to determine from the facts available whether this was the primary affection, or whether it was secondary to the disease of the other elements, or, thirdly, if all the cells of the cortex were equally affected from the beginning. From the study of other lesions I am inclined to agree with those who regard the Purkinje cells as the most vulnerable elements of the cerebellum, but this is no proof that in a case of primary cortical degeneration these would be the first affected.

The degeneration of the myelinated fibres of the folia and white matter of the cerebellum may be most naturally regarded as the result of the degeneration of their cells of origin in the cortex, but it was not complete in any portion of the cerebellum, despite the great intensity of the cortical disease. This may be explained by the fact that the fibres which spring from the cells of the cortex are intermixed with the afferent fibres which enter the cerebellum through its inferior and middle peduncles; and the majority of these, in fact all except the olivo-cerebellar fibres, were normal.

Further, the efferent systems from, and the association fibres between, the central nuclei were intact, as these were unaffected by the disease. It seems then probable that the only myelinated fibres which had disappeared were those which take origin in the cortex and connect this with the central nuclei, or pass as arcuate fibres between the folia.

The proliferation of the neuroglia and the sclerosis of the ground tissue were the natural result of the degeneration and disappearance of the functional elements of the cerebellum; it was consequently most marked where the degeneration was greatest. But the form and degree of this sclerosis in the different layers of the cortex are dependent on the normal amount and arrangement of the neuroglia in its different portions. The granular layer is remarkably poor in neuroglia tissue, as Weigert pointed out [15], and consequently there is rarely a striking amount of sclerosis within it, no matter how advanced its degeneration. In the molecular layer, where it is also scanty, the glia is arranged in radial fibrils (Bergmann's) which had become thickened, and consequently gave this layer a striate appearance. The cells of origin of these radial fibrils lie in the Purkinje layer, and this, which is the portion of the cortex best supplied with neuroglia, contains many neuroglia cells, which also supply fibrils to itself. Owing to the proliferation of these cells, an abnormal belt of nuclei is found in this layer, and is in many places a striking feature in the cross-section of the folia. The prominent dense strands of neuroglia which have been described in this layer have arisen by the proliferation of these cells. The sclerosis of the white matter was secondary to the degeneration of its myelinated fibres; it is proportional to the degree of this degeneration in the different regions, and is consequently greatest in the medulla of the folia and in the dorsal periphery of the white matter of the lateral lobes.

The presence of this complementary neuroglial sclerosis is positive evidence of the degenerative nature of the cerebellar disease, and excludes the possibility of regarding the condition as an arrest of development or agenesis; it is definite evidence that tissue elements have disappeared.

Emphasis is laid on this point, as Thomas, in describing cases of degeneration of the cerebellar systems [12], uses the absence of marked neuroglial sclerosis as an argument in favour of the primary affection of the nervous elements. If his intention was to argue that the presence of sclerosis would have pointed to the degeneration of the nervous tissue being secondary to neuroglial overgrowth or vascular lesions, the same argument would have to be extended to all degenerations of the nervous system; this is an untenable position.

The degeneration and sclerosis of the olives and the disappearance of the olivo-cerebellar fibres must be now considered. Atrophy of an olive after lesions of the opposite lateral lobe of the cerebellum has been frequently observed since it was first described by Meynert. The final changes after experimental lesions of the cerebellum are similar to those in the case described above, namely, atrophy and eventual disappearance of the cells, and wasting of the white matter of the hilum of the olive and of some of the transverse fibres of the interolivary layer. These changes have been variously interpreted. For a time they were taken as evidence that the olivo-cerebellar fibres are cerebellofugal, and consequently degenerate when their cells of origin in the cerebellum are destroyed; but this failed to explain the fact that changes are found in the olivary cells very early after cerebellar lesions, and that the affection of the olives is much more similar to the condition of Gudden atrophy—that is, cellular regression occurring in grey matter when its efferent fibres are destroyed—than to the changes which occur in nuclei when their afferent fibres have been severed; then the chief incidence of the change falls on the interstitial substance in which these afferent fibres arborise. Now, however, it is known from the investigations of Lewandowsky [7], van Gehuchten [4], and others by Marchi's method that the connection of the olives with the cerebellum is entirely cerebellopetal. Thomas [12], also by Marchi's method, has found fine black granules in an olive after ablation of the opposite lateral lobe of the cerebellum

although no degeneration was visible in the fibres which pass between them; this fact agrees with van Gehuchten's observation [5] that the regressive changes in the central end of a divided fibre commence in the neighbourhood of its trophic cell. The examination of the inferior olives at different periods after lesions of the cerebellum leaves in my mind no doubt that the essential change is swelling and tigrolysis and the eventual disappearance of the cells of the olive contralateral to the cerebellar lesion, in fact, the typical cell change known as "reaction à distance."

On the other hand, pronounced alterations of quite different character have been frequently observed in the olives after degeneration of their chief afferent bundle, the central tegmental tract. In Thomas' case [14], for instance, the olive was apparently hypertrophied owing to the swelling of its ground tissue, and, presumably secondary to this, there was a diminution in the number of its cells and of the volume of those which remained.

It must be therefore concluded that the pathological changes found in the olives associated with cerebellar disease are due to involvement of the olivofugal fibres in the lesion. Although in my case these were not involved in any gross lesion, it does not seem necessary to assume that the atrophy of the olives was primary. The terminations of the olivo-cerebellar fibres in the cerebellar cortex may have been involved in the sclerosis, or the lesion may have been physiological rather than anatomical, the degeneration being due to disturbance of functional connections, and extending over a synapse into the second neurone. This has been freqnently observed with experimental as well as with pathological lesions, especially in young animals. The great tendency of the inferior olives to undergo atrophy secondary to cerebellar disease is best illustrated by the fact that atrophic changes have been found in them in cases of atrophy of one half of the cerebellum secondary to lesions of the opposite cerebral hemisphere. In several of these cases, as in those of Cornelius [2] and Reitsema [10], in which any direct influence of the cerebral lesion could be excluded, and in which the cerebellar atrophy was

uncomplicated by inflammatory or reactionary processes, the alterations in the olives were practically identical with those which are found in my case of primary cerebellar disease.

In contrast to the condition of the olivo-cerebellar fibres, no pathological change was found in any of the other afferent systems of the cerebellum.

Of the predisposing factors and of the etiology of the degeneration of the nervous elements of the cerebellar cortex nothing more can be asserted than of other forms of family dystrophy. The clinical histories of the cases bring out two important points: in the first place the family nature of the disease, and secondly, the existence of congenital or developmental anomalies in the family—the scanty amount of hair, and the small size of the tonsils, and of the penis and testicles in the males. These anomalies make it probable that the selective disease of the cerebellar cortex was primarily due to an hereditary defective vital endurance, or a tendency to degeneration of this tissue, which began to fail from the middle of adult life.

The chief interest of these cases from the anatomical and physiological points of view lies in the nature of the cerebellar disease. The cerebellopetal tracts, by which the cerebellum receives impulses from the sensory structures of different parts of the body through the intermediation of other parts of the central nervous system, were intact, except the olivo-cerebellar fibres and their cells of origin, the degeneration of which was only secondary to the cerebellar disease. Recent investigations make it probable that all afferent fibres terminate in the cortex of the cerebellum, and that none end in the intrinsic nuclei.

Clarke and Horsley [1] have shown that the efferent fibres of the cerebellum, on the other hand, spring from its central nuclei only, and both these and their efferent fibres were normal in the case which is reported here.

The degeneration was thus limited to the cortex, or the receptive part of the cerebellum, and to the fibres which arise in it. The latter belong to two classes: the projection fibres, represented by the axones of the Purkinje cells,

which connect the cortex with the intrinsic nuclei, and the association fibres which pass between neighbouring or more distant folia.

As the cerebellum may be regarded as a massive afferent recipient organ (Horsley), or the head-ganglion of the proprio-ceptive system (Sherrington), it must be assumed that the chief function of its cortex or receptive portion, in which afferent impulses below the realm of consciousness, from many regions of the body and consequently different in nature converge, is to sort out and coördinate the relatively crude impulses it receives and transmit them in more compact and more highly organised arrangements to the deep nuclei which are in direct efferent connection with the other parts of the central nervous system. According to this view, for instance, the labyrinthine impressions and afferent impulses from the trunk and lower limbs, both of which are necessary for equilibration and the direction and coördination of the movements of progression, are associated and conjoined in the cerebellar cortex and nuclei. The great excess of the afferent over the efferent systems of the cerebellum, and the relative immensity of the cortex in comparison with the intrinsic nuclei, also suggest that the chief function of the cerebellum as a whole is to concentrate and organise the different forms of afferent impulses outside consciousness for the service of other parts of the nervous system.

Cerebellar disease of the type recorded here, which may be defined as an affection of the cerebellum on its afferent side, must then, if complete, lead to total loss of its functions, as it has destroyed its receptive portion and the links which connect this with the efferent centres; the latter, it must be assumed, remain inactive when deprived of all afferent impulses, or are at the most capable only of irregular and uncontrolled activity.

Although the clinical notes of these cases were not complete—the greater part of them were written twenty years ago—they are sufficient to allow us to correlate the structural changes with the symptoms which were observed during life. On analysis it is seen that all these symptoms

were due to disturbance of the coördination, or to the asynergia of muscular activity in the different parts of the body. As this incoördination was not associated with any defect of power or loss of any form of sensation, it seems directly referable to disease of some portion of the cerebellar system, including under this term not merely the cerebellum itself, but also its afferent and efferent tracts. Further, the nature of the incoördination, or rather the asynergia of the movements, is such as is found in subacute or stationary cerebellar disease.

The staggering and drunken gait and the defect in equilibration, unaffected by deprivation of the aid of vision, were evidently the result of this muscular incoördination; and the ataxia of the upper extremities, which was the next most prominent symptom, was undoubtedly due to the same cause; it also is a well-recognised symptom of cerebellar disease. The peculiar affection of articulation needs more discussion; the occurrence of incoördination in the bilaterally represented movements is not so well recognised in cerebellar disease owing to the fact that it rarely occurs in the forms of cerebellar disease which are most frequently met with—that is, in focal lesions. In cerebral disease, too, disturbance of articulation is rarely the result of unilateral lesions, though it may be affected out of all proportion to movements unilaterally represented by bilateral destruction of the motor centres or their efferent fibres (pseudo-bulbar palsy). Similarly the delicately coördinated mechanism of speech is rarely affected by even extensive unilateral lesions of the cerebellum, but its disturbance may be a prominent symptom when the lesion involves both sides of the cerebellum. In one case, after removal of an extensive infiltrating glioma of the cerebellum, I observed articulation become slow, jerky and explosive, with loss of the natural intonation of words, owing to incoördination of the muscles of articulation and phonation. It has persisted, with but little recovery, for now over three years.

Nystagmus is such a constant symptom in cerebellar disease that it needs no discussion; the irregular nystagmoid jerkings which complicated the volitional movements

of the eyes in these cases must be interpreted as a disturbance of their coördination.

The position of these cases among the cerebellar diseases need not be discussed fully here; the whole subject is treated in a review in the present number of this Journal. I have not been able to find references to any absolutely similar cases in the literature. Those reported by Fraser [3], which were also familial, were probably the most nearly similar, but in them the symptoms were present from early childhood, and the anatomical examination of the nervous system was not sufficient to allow us to exclude with absolute certainty disease of other parts; in fact, there was optic atrophy in addition to the cerebellar disease.

In the case published by Thomas [13] the symptoms resembled those of the cases recorded here, but the disease was not familial and the affection of the cerebellar cortex was much slighter.

In all the other cases of primary progressive cerebellar degeneration other portions of the central nervous system were also affected, or the pathological changes of the functional tissue were secondary to vascular or interstitial disease.

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PLATE I.

A

B

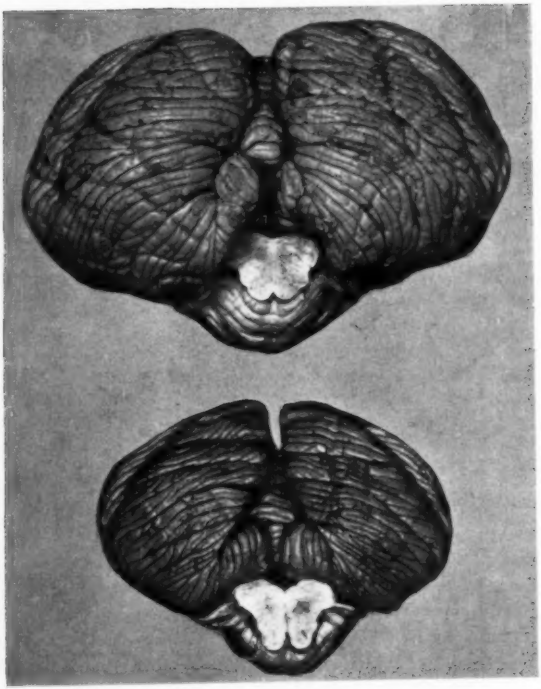
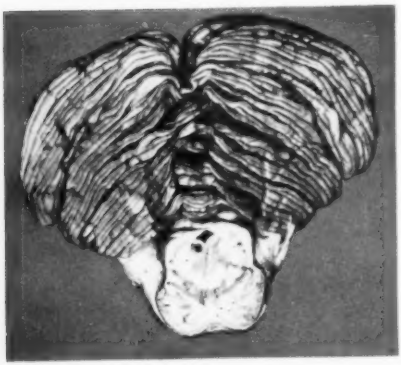


FIG. 1.



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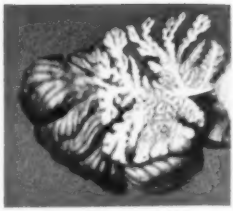

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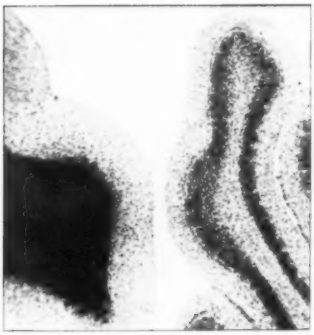


FIG. 4.

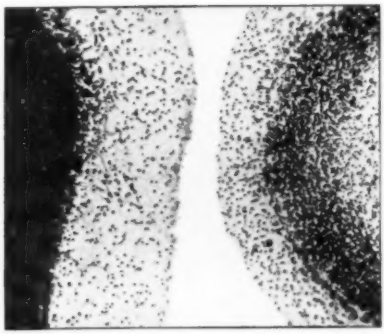
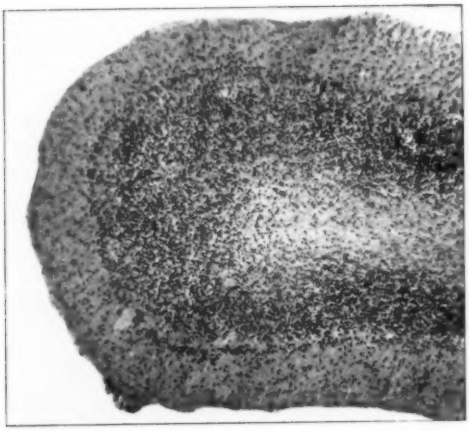


FIG. 5.



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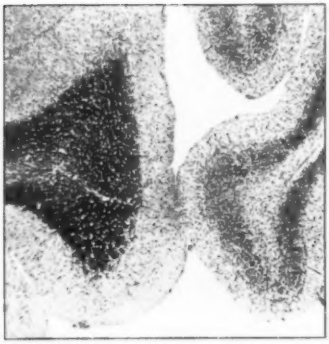

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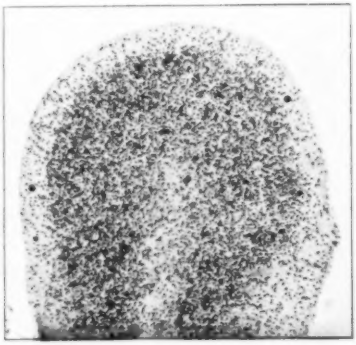

FIG. 7.

PLATE III.

FIG. 9.

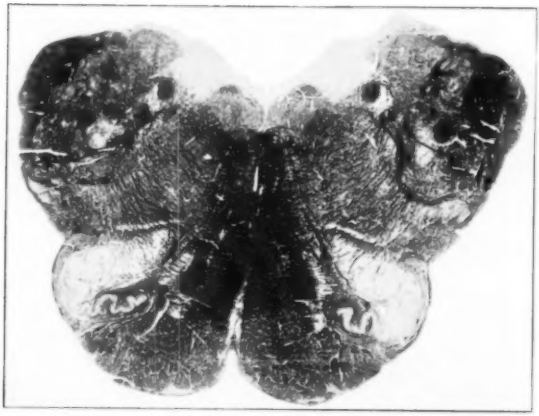

FIG. 10.

Description of Figures.

Fig. 1.

B, cerebellum from Case 4. *A*, cerebellum of a brain in which the cerebrum was of the same weight as that of Case 4. The two were photographed with the same magnification to show the small size but normal shape of the atrophied cerebellum.

Fig. 2.

The superior surface of the cerebellum, to show the narrow and wasted folia and the abnormally wide sulci.

Fig. 3.

The cerebellum cut in the median sagittal plane of the vermis. It shows the striking atrophy of the folia of the vermis, those of the uvula, nodule, and of the central lobule being the least affected.

Fig. 4.

Photograph of a relatively normal and of a wasted folium, separated by the posterior superior sulcus. (This and the two following figures were selected to show the contrast between the relatively normal and the severely affected folia.)

Fig. 5.

Ditto. Note the marked atrophy of the molecular and granular layers, the disappearance of the Purkinje cells, and the presence of an abnormal belt of nuclei in the intermediate layer of the folium to the right of the figure.

Fig. 6.

Folia from the tonsil; that to the left is relatively normal, but it does not contain many Purkinje cells.

Fig. 7.

The tip of a folium from the flocculus, showing great atrophy and disintegration of the cortex, increase of glial nuclei in the molecular layer, and the presence of corpora amylacea.

Fig. 8.

Section of an atrophied folium to show the prominent belt of neuroglia nuclei in the intermediate (Purkinje) layer.

Fig. 9.

Weigert-Pal section through one half of the cerebellum and the pons. There is marked degeneration of the myelinated fibres of the folia and considerable rarefication of the white matter of the lateral lobe; the dentate nucleus and the superior cerebellar peduncle are normal, and the commissures of the vermis and the fibræ semicirculares stain better than the rest of the white matter. There is no degeneration in the pons.

Fig. 10.

A Weigert-Pal section of the medulla. It shows the small size of the corpora restiformia and the degeneration of the lateral halves and of the whole of the dorsal layers of the olives.

THE PRECISE DIAGNOSTIC VALUE OF ALLOCHIRIA.

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I.—Introduction and Definition.

In the quarter of a century that has elapsed since Obersteiner gave the first clear description of this condition, remarkably little interest has been displayed in it by either neurologists or psychologists. The literature on the subject is sparse and the number of cases that have been recorded is astonishingly few; in less than half a dozen of these has any exact account of the patient's sensory state been given, the symptom being, as a rule, just briefly mentioned, and in only one case has the condition been at all fully described. In a large number of standard text-books, both on general medicine, such as those of Clifford Allbutt, Gibson, Fagge and Pye-Smith, Allchin, and the "Twentieth Century Practice of Medicine," and on neurology, such as those of Allen Starr, Sachs, Dercum, De Fleury, the symptom is not even referred to. In the majority of those that do mention it, quotations from which will presently be given, the writer is content with a definition, or perhaps adds that the symptom occurs in a number of diverse affections, of which tabes and hysteria are the most frequent. No text-book ascribes any value in diagnosis to its presence, with the sole exception of Musser's "Medical Diagnosis" [106], in which it is merely stated that allochiria when general should be regarded as a stigma of hysteria, but when local usually occurs with organic disease of the spinal cord.

The reasons for this lack of interest are perhaps as follows: On the practical side the symptom has been thought to be devoid of value in clinical diagnosis, on account of the multiplicity of the diseases in which it has been observed. On the theoretical side it has similarly been denied interest largely because a simple mechanical explanation, which will presently be criticised, has been widely accepted, so that it would appear as if there remained no further problem to be elucidated. It is my opinion that on both these sides there is scope for a revision of the views at present held on the subject and also for a further elaboration of our knowledge as to the significance of the phenomenon. In the present paper I propose to deal with

only the former of these two sides of the subject, for a discussion of the pathogenesis of the conditions concerned, raising as it does some of the most complex questions in psychology, would lead one beyond the scope of a single article. The second side of the subject will be dealt with in a succeeding article.

In the first place it will be necessary fully to consider the various definitions that have been offered; for lack of precision in this respect has led in the past to considerable obscurity. There has been no clearer or more satisfactory definition suggested up to the present than that originally given by Obersteiner [107] when he said that he applied the term allochiria to a condition in which, "though the sensibility is retained more or less completely, the patient is not clear, or is frequently, if not constantly, in error as to which side of the body has been touched." He adds: "The power of localisation is retained as to details, whilst doubt or error exists as to the side touched, the irritation being commonly referred to the corresponding part of the other limb." The essential feature of the condition, according to this definition, is that *in the patient's mind there exists doubt or error as to which side of the body is touched.*

Unfortunately, in later descriptions of the condition attention has almost always been attracted to what is the more striking though probably the less important part of the definition, namely, the feature that the stimulus is commonly referred to the corresponding part of the other limb. On account of its unconsciously biasing the writer to one particular hypothesis as to the explanation of allochiria, this point of view is largely responsible for the sterility of our conceptions of the condition; the following instances show how almost universally it is entertained. Gowers and James Taylor in their text-book [54] call allochiria "a condition in which an impression on one part has been referred to the corresponding place on the opposite side of the body," and in Gibson's "Text-book of Medicine" [55] they say it is present when "a prick or touch on one leg is referred to the other." Ferrier [43] terms allochiria "a form of perversion of sensibility, characterised by an erroneous reference of

sensory impressions to the corresponding part of the other side of the body." Church [28] says that "sometimes the impression is felt on the opposite side of the body at a symmetrical spot—allocheiria." Gay [48] says that it "consists of a perversion of common sensory impressions to such an extent that they are localised to an exactly corresponding area on the opposite side of the body." Musser [106] says that it is "a term employed to describe the reference of a sensory stimulus to the corresponding location on the opposite side of the body." Judson Bury [26] says that "a patient may refer an impression on one side to a corresponding place on the opposite side of the body—then the condition is named allocheiria." Osler [114], speaking of tabes, says that "if the patient is pricked on one limb he may say that he feels it on the other (allocheiria)." Frederick Taylor [127] states that "a touch may be felt at the corresponding part of the opposite side of the body (allochiria)." Pierre Marie, writing in the Charcot-Bouchard-Brissaud "Traité de Médecine" [96], says: "Parfois ils localisent la piqûre au point correspondant du membre opposé, c'est ce qu'on a appelé l'allochirie." Dejerine and Thomas, writing in the Brouardel-Gilbert "Traité de Médecine" [30] say: "Exceptionnellement la sensation est perçue sur l'autre membre; c'est de l'allochirie." Brown-Séquard [22] speaks of it as "consistant en ce que le malade croit que l'impression sensitive a été faite sur un côté du corps alors qu'elle l'a été sur l'autre côté." Longuet [92], in his review of the subject, describes allochiria as follows: "Si l'on frôle la peau de la cuisse, du mollet ou de la plante du pied de certains malades, le contact, parfaitement ressenti par le patient, est rapporté par lui, non au membre touché, mais aux points exactement correspondants du membre symétrique." Gellé [51] writes: "L'allochirie consiste dans la perception d'une sensation dans le côté du corps opposé au point où l'excitation a lieu." Mlle. Lapidous, in her thesis on allochiria [85], writes: "Il s'agit d'un trouble dans la localisation des mouvements et des sensations qui fait que le malade rapporte à un côté une sensation partie du côté opposé du corps." Victor Henri [61] writes: "Le malade

commet une erreur de côté; le contact d'un point d'un membre droit est rapporté au point correspondant du membre gauche et réciproquement." Strümpell, in his text-book [126], says: "Die Allocheirie besteht darin, dass ein Hautreiz nicht an der gereizten, sondern an der entsprechenden Stelle auf der anderen Körperhälfte empfunden wird." von Leyden and Goldscheider, in Nothnagel's "Specielle Pathologie und Therapie" [89], call allochiria "ein seltenes und wenig verwerthbares Symptom, darin bestehend, dass ein Reiz, welchen man an einer Extremität des Kranken applicirt, von diesem an der entsprechenden Stelle der anderen Extremität localisirt wird." Huber defines it [66]: "Dass die Kranken die Empfindung eines Reizes nicht in die gereizte Extremitäten, sondern in die entsprechende der anderen Seite verlegen." Weiss calls it [130] "eine Störung der Localisation welche darin besteht, dass die Wahrnehmung einer Empfindung von der gereizten Stelle auf die entsprechende der anderen Körperseite verlegt wird." Hoffmann writes [64]: "Wenn Kranke einen Reiz nicht an der tatsächlich gereizten Hautstelle sondern denselben nach der correspondirenden Stelle der anderseitigen Gliedmassen localisiren, so bezeichnet man diese eigentümliche Anomalie als Allochirie." Morselli [100] defines allochiria as "un fenomeno singolare del riferire la sensazione tatile al punto opposto, ma simmetrico, nel quale veniva toccato."

Even those authors who quote Obersteiner's definition in full usually proceed to consider allochiria purely from the limited point of view indicated above. Thus Blocq, who writes the article on the subject in Richet's "Dictionnaire de Physiologie" [8], first quotes Obersteiner's definition in full and then adds: "Ce signe consiste, en somme, dans le fait de rapporter à une région plus ou moins symétrique du membre de l'autre côté." Bosc, in his monograph on the subject [11], goes so far as sharply to separate the cases in which doubt in the patient's mind as to the side touched is the prominent abnormal feature from those in which deflection of sensation to the wrong side occurs. He calls the former false or pseudo-allochiria, and defines true allochiria [12] as

the condition in which "un malade rapporte à un côté une sensation partie du côté du corps opposé."

It will be seen that, although the above definitions differ considerably on such points as the nature of the stimulus concerned and the degree of exactness in the symmetry between the site of the stimulus and the seat of its localisation, yet they all agree in one thing, namely, that the essential feature in allochiria is the deflection of a sensation to the wrong side of the body. In none of these definitions is any stress laid on the state of the patient's knowledge of right, or left-sidedness, an aspect of the problem which is, in fact, of fundamental importance. In other words, we have no hint so far of the symptom being anything more than a bizarre error in localisation. The position that this point of view logically ends in is reached by Buzzard [27] when he defines allochiria as a condition in which "a point of contact is referred, not to its true locality, but either to some part of the opposite limb or to a distant part of the same member." Here we leave altogether the point on which Obersteiner laid such stress, namely, that there is in allochiria no defect in vertical localisation but merely a confusion in the patient's mind between the opposite sides of the body, and come to look upon the symptom as simply any form of bad mistake in localisation. I shall have later to insist on the significance of this distinction made by Obersteiner, which at first sight seems so unimportant, for I am convinced that the relative sterility of most discussions on the subject is largely due to its having been overlooked. In the literature I have found only three passages outside of Janet's writings in which the above distinction is noted. The first is in Da Costa's "Medical Diagnosis" [29], and runs thus: "Allochiria is a form of perverted sensibility, which may or may not be associated with anæsthesia, and consists in the sensibility being more or less perfect, while there is doubt as to the side touched; indeed, the touch is commonly felt at a corresponding part of the other limb." The second is one in which Mott quotes Obersteiner's definition in full without subsequently modifying it [102]. The third is the definition given by Brécy in the recently appeared "Pratique

Médico-Chirurgicale" [19], in which he says: "Les malades ne reconnaissent plus sur quelle moitié du corps a porté l'excitation, ou bien la localisent en un point plus ou moins symétrique du côté opposé."

On the few occasions in which writers have thought of allochiria as a confusion between the opposite sides of the body rather than as a defect in localisation, they have, curiously enough, overlooked the remaining part of Obersteiner's definition, namely, that the confusion, when present, is in the patient's mind. It has thus come about that almost every symptom or physical sign in neurology that can in any sense of the word be "referred" to the opposite side of the body has been called allochiria, without any further analysis being made of the exact condition present. This to a great extent accounts for the multiplicity of both the varieties and causes of allochiria that have been described, and naturally has tended to confuse and obscure the true picture of the condition. It will be seen on investigation that many of these various affections described as allochiria have hardly anything in common with the condition described by Obersteiner and defined above. To sum up the cardinal features of the condition so defined, there is in the patient's mind doubt or error as to the side touched, whilst sensibility, including the power of localisation, is otherwise retained. Even though only those cases that come strictly under this definition of Obersteiner's be given the name allochiria, there remain, as will presently be pointed out, a sufficiently great number of varieties of the condition.

II.—Review of Published Cases.

Four previous reviews of the literature on the subject have been made: by Longuet in 1884 [92], Weiss in 1891 [132], by Bosc in 1892 [13], and by Determann in 1900 [31]. The last mentioned, who adds but three cases to those collected by Weiss, states that altogether only a little over a dozen cases of all varieties are on record. A more fortunate search, however, has enabled me to read the accounts of some seventy-six cases to which the term allochiria has been applied, and I will briefly consider them

under the various groups into which they enter. Allochiria has been described as occurring in peripheral nerve lesions, hemiplegia, disseminate sclerosis, tabes dorsalis, unilateral injury to the spinal cord, Menière's syndrome, hysteria, symmetrical gangrene; and in connection with touch, pain, the "muscle sense," the temperature sense, sight, smell, taste, hearing, and the electrical reactions. As stated above, many of the cases would be denied the designation allochiria were the original definition adhered to.

(1) *Electromotor Allochiria.*

In 1892 Weiss [133] applied this term to a condition in which "auf elektrische Reize nicht die von Strome durchflossenen Muskeln oder Muskelgruppen, sondern die correspondirenden, nicht erregten der anderen Körperseite mit Zusammenziehung antworten." The expression, however, is usually applied less strictly than is here indicated, and is made to include conditions in which the stimulated muscle responds as well as the contralateral muscle; these Petrina and Senator had previously spoken of as "crossed electrical reflexes." The condition had been previously described on many occasions, three of which are mentioned by Weiss, the first being when Remak mentioned it in 1858 [117] in a case of tabes, under the designation "reflex electrical movements." Similar findings have been recorded in nuclear facial paralysis by Benedikt [1] and Petrina [115], in infranuclear facial paralysis by Brenner [20] and Senator [121], in supranuclear facial paralysis by Hoffmann [65], in traumatic neuritis of the external popliteal nerve by Meyer [97], in myelitis by Gräupner [56], in probable disseminate sclerosis by Fischer [44], in hysteria by Féré [41], and Binet and Féré [4]; they have also been noted by Erb [40]; Weiss calls his case one of symmetrical gangrene [135], but from the atrophy of the small hand muscles, Charcot's joints and other trophic disorders, characteristic sensory changes, and course of the disease, it reads very much like a case of syringomyelia.

The phenomenon may be shown to faradism only (Weiss, Petrina), galvanism only (Remak, Gräupner, Fischer), or to

both faradism and galvanism (Senator, Hoffmann). In cases in which the reaction of degeneration is present the contralateral contraction may similarly show polar changes, indicating a specific action *via* the central nervous system (Gräupner). It has been observed in the face (Brenner, Petrina, Benedikt, Hoffmann, Senator), in the lower limbs (Remak, Meyer, Gräupner, Fischer), and in the upper limbs (Weiss). The contralateral muscles may contract equally and simultaneously with the homolateral muscles, to a less degree, to a greater degree, or they alone may contract. In the last instance the homolateral muscles may contract only with a stronger current than is necessary to evoke contralateral contraction, and in this case their contraction will be less marked than that on the opposite side (Weiss). In nearly all the above cases the stimulus was applied on the affected side, but in Hoffmann's case stimulus of the healthy facial muscle caused contraction of the opposite and paresed facial muscles with a current so weak that the healthy facial did not react. The distant contraction is usually of localised muscle groups, but in Meyer's case it was more general. The clinical significance of the phenomenon is still doubtful. There was for some years a controversy as to whether it always indicated a nuclear lesion, as maintained by Benedikt [2], or whether it could occur also in infranuclear lesions, as was finally demonstrated by Brenner [21]. The majority of the cases, and possibly all, occur with lesions of the lower efferent neurone, other electrical changes being frequently present as well. It is possible that it occurs with organic affections only, for although five out of the above seventeen cases (one described by Féré and four by Binet and Féré) were cases of hysteria, the observations are open to the following criticism. They were published twenty years ago before the importance of the effects of suggestion was recognised, and were studied under the influence of Charcot's teachings on the subject before these were discredited. All the cases had previously been experimented upon and contralateral contraction of muscles induced by mere pressure or touch. Phenomena which are now known to be

the effect of suggestion, such as Charcot's neuro-muscular hyperexcitability, somnambulism from pressure on the vertex, &c., were described in the same cases and their significance erroneously interpreted. The fact that pressure on one forearm caused movement of the opposite forearm forces one to regard with grave suspicion such an occurrence as contraction of the opposite forearm muscles from faradism of the forearm. It is therefore almost certain that these cases should be placed in a completely different category from the twelve organic cases mentioned above.

The origin of the phenomenon in the organic cases is extremely obscure. The fact that contraction of the arm may occur in hemiplegia when the leg is stimulated, and the specific dissociation seen sometimes with galvanism, point strongly to an action *via* the central nervous system. This specific dissociation was mentioned above as occurring in Gräupner's case. Again in Braun's case of hemiplegia, probably of thrombotic origin [18], stimulation of the external popliteal nerve by galvanism caused a marked increase in the contracture of the homolateral arm; this had previously been described by Remak [118], but the interesting feature of Braun's case was that making and breaking the current, applied in this instance to the internal popliteal nerve, regularly caused abduction and adduction of the arm so that what he described as a pendulum movement was set up. What seems to be quite unexplained, however, is the nature of the nervous change that permits an electrical stimulus to be conducted so readily and to act at a distance when the local action is in abeyance. That there is some such special change in the nerve conductivity is indicated, as Weiss hints, by the peculiar nature of the distant contraction, such as the delay before its appearance, the lasting tetanic contracture that may ensue, &c.; we have clearly to do with something more than a mere reflex movement.

Whatever may be the final explanation of this interesting phenomenon, enough has been said to make it evident that the whole subject is an entirely different one from that of allochiria as defined above. The central fact is that an

electrical stimulus may manifest its effect at a distant part of the nervous system; this distant part may be on the same side of the body or on the opposite side; naturally it is more often on the opposite side, because the representations of corresponding contralateral limbs in the spinal cord are nearer to each other than are those of homolateral limbs. This fact, however, has nothing whatever to do with the confusion of the two sides that occurs in the patient's mind when allochiria is present.

(2) *Motor Allochiria.*

The contralateral muscular contraction induced on striking or even touching one limb, described by Trapieznikow [128], Dumontpallier [38], and Binet and Féré [5], will be discussed later in connection with allokinesia.

(3) *Reflex Allochiria.*

Weiss [133] gave this name to the condition present in Ferrier's case [43], in which stimulation of the sole of the foot or of the inner part of the thigh evoked the corresponding reflex on the opposite side only. The same occurrence had been described also by Binet and Féré in connection with the knee-jerks [6.]

Since this date, however, bilateral or contralateral evocation of reflexes has become a well-recognised phenomenon in cases when their activity is exaggerated, and in one instance —the crossed adductor jerk—it is especially common. There is, of course, no possible resemblance to allochiria in the bilateral cases; and even when the reflex is elicited only on the contralateral side we have merely to instance the analogy of the cases in which the direct reaction of the pupil to light is abolished while the consensual reaction is preserved—a condition which no one would think of calling allochiria—to perceive the fundamental dissimilarity of the condition from allochiria.

(4) *Allochiria in connection with the Special Senses.*

(*a*) *Auditory.*

Three cases have been recorded in connection with the sense of hearing. The first, that of Gellé, was published in

1888 under the title of "Auditive Allochiria in Menière's Disease" [53]. The patient had auditory vertigo due to chronic otitis, and the left ear showed marked hyperæsthesia and hyperexcitability. A bruit that Gellé heard with a stethoscope placed over the right carotid was heard by the patient in the left ear. There seems no reason why such a condition should be called allochiria, for not only is our knowledge very slight as to what such an arterial bruit corresponds with in fact—it is conceivable that the physical signs described might co-exist even with a condition causing a bruit to be really produced on the left side—but even were the observation to be accepted as significant it would be merely an extension of the usual Rinné test. It is not very rare for a tuning-fork to be heard constantly by one ear, on whichever side of the skull it may be placed.

The other two observations were recorded recently by Bonnier [10], one of them in considerable detail, under the title "Allochirie Auriculaire." In these cases a plug of cerumen in one meatus caused a series of symptoms, including pain, deafness, vertigo with unilateral forced movements, &c., which were referred to the opposite ear. The symptoms ceased after the removal of the wax. Discussion of these cases will be reserved until later.

(*b*) *Visual.*

Two cases of allochiria in connection with vision have been recorded, both in 1888. One was recorded by Féré under the title "Optic Allochiria," but I have not been able to read the original account [42]. The case is said[1] to have occurred in a female hysteric in whom the visual impression received by the right open eye was regularly referred to the left eye, the patient maintaining that she perceived the impression with the left eye, which in fact was shut.

In the same year a similar case was fully described by Magnin, under the title "Visual Allochiria" [94]. The patient, also a female hysteric, had total and complete left

[1] I use this phrase advisedly, for there are certain inconsistencies in the cited accounts of the case that justify some scepticism as to the accuracy of the descriptions.

hemianæsthesia. This included on the same side the conjunctiva, cornea, auditory meatus, and the mucous membrane of the mouth and pharynx. Smell, taste and hearing were abolished on the left side, and on this side there was also absolute achromatopsia and almost complete amaurosis. During hypnosis, when both eyes were open, but their fields separated by the use of a screen, a coloured object held in front of the left eye was recognised, and the patient maintained that she saw the colour with the right eye.

(*c*) *Gustatory.*

In Ferrier's case [43] a substance placed on one side of the tongue was said to have been tasted on the opposite side, but as touches on that side of the tongue were also referred to the opposite side it is difficult to see how one can in this case dissociate the reference of taste from that of touch.

(5) *Sensory Allochiria.*

We now come to the cases described as sensory allochiria, forty-one in number. I propose to divide these into two groups, which are fundamentally different in their symptomatology and pathogeny. These two groups I shall call for the present (1) alloæsthesia or false allochiria, and (2) dyschiria, including true allochiria, respectively, reserving until later the description of their distinguishing features. Obersteiner did not distinguish between them, nor so far as I know has any other writer, although only the second group conforms to the definition he laid down.

First must be mentioned a condition that has been called allochiria by Longuet [92], but which really enters into neither of the above groups. This is the peculiar diffuse burning pain, termed causalgia by Weir Mitchell [98], that occurs after section of a peripheral nerve, and which may radiate from one limb so widely as to include even the opposite side of the body in its range. Cases of this nature are mentioned by Hutchinson [68], Pirogoff [116], Weir Mitchell [98], and others. While it is probable that false allochiria may occur in bilateral affections of peripheral

nerves, such as multiple neuritis, although no such case has yet been recorded, still it is clear that the condition above mentioned is in no sense related to either variety of allochiria. The mere fact that a lesion may produce a manifestation at distant points, and that some of these distant points may be on the other side of the body from the lesion, has little in common with allochiria. Morselli [100] described under the incorrect title of allochiria a curious case of Jacksonian epilepsy in which a stimulus applied to the inside of the cheek was referred to the outside, and *vice versâ ;* a touch on the index finger was referred to the thumb. From the brief description given it is difficult to know how the case should be classified.

Certain of the recorded cases of allochiria are described so incompletely that it is impossible to determine which group they belong to. Such are three recorded by von Leyden [90] and Obersteiner [108]. Cases have also been observed by Musser [106], Schiff, Charcot, Van Deen [120], accounts of which I have not been able to find.

The number of probable cases of alloæsthesia or false allochiria on record is eleven. Five of these, published by Brown-Séquard [24], Hertzberg [63], Fischer [45], Obersteiner [109], and Huber [66], almost certainly belong to this category, while the other six, published by Brown-Séquard [25], von Leyden [91], Fischer [46], Obersteiner [110], Determann [32], and Spearman [123], probably do also, as far as one can tell from reading the description of them.

We come last to the cases that enter into the group of true allochiria. They are thirty-one in number, of which two can be classed with only a moderate degree of probability. These two were published, one by Ladame [84] in 1865, the other by Bikeles [3] in 1899. The former was a case of pontine tumour, the latter of a bullet-wound in the motor area. The symptom is only briefly mentioned in both cases.

The other twenty-nine cases were published in this order, one by Obersteiner [111] in 1881, one each by Ferrier [43]

and Dobie [37] in 1882, one by Hammond [57] in 1883, two by Magnin [95] and one by Féré [41] in 1888, one by Pierre Janet [69] in 1890, one by Weiss [130] in 1891, two by Bosc [14] and one by Kerr [83] in 1892, one by Gay [50] in 1893, eleven by Sollier [122] in 1897, one by Lapidous [86] in 1899, three by Trapieznikow [128] in 1901, and one by Sabrazès and Bousquet [119] in 1905. Of these twenty-nine, twenty-two were published as being straightforward cases of hysteria and nothing else; these comprise eleven by Sollier, two each by Magnin and Trapieznikow, and one each by Obersteiner, Dobie, Féré, Janet, Bosc, Kerr, and Lapidous. In two of these cases the symptom occurred only during hypnosis, one being published by Magnin, the other by Bosc. Two more were published as being cases of hysteria, combined, one certainly and the other possibly, with organic disease. The former (Weiss's case) was one of tabes certainly combined with hysteria. The other (Sabrazès and Bousquet's case) was one of undoubted hysteria, which, however, the authors suspect may have been superadded to some organic affection such as disseminate sclerosis; there was, however, no evidence of this, and it was clear, as the authors state, that the hysteria that was certainly present could have accounted for all the symptoms. Of the remaining five, two were observed a quarter of a century ago, at a time when our knowledge of the more severe forms of traumatic hysteria was extremely small. Reading the descriptions of them in the light of modern studies such as that of Sidis's well-known Hanna case, &c., one can say almost with certainty that in both instances the diagnosis of traumatic hysteria would have been made to-day. In the one case, that of Hammond, the condition was thought to be lateral sclerosis; in the other, that of Ferrier, no diagnosis was given. Of the three remaining cases, in one, published by Trapieznikow, no details are given in the German translation accessible to me beyond the statement that the patient had vertebral ankylosis; the writer, however, comes to the conclusion that the symptom of allochiria is always of psychical origin, so we may suppose that some psychoneurosis was present in this case. In the other two cases

an organic affection of the nervous system was present, but in both there was reason to suspect the presence of hysteria. In Gay's case of diphtheritic paralysis in a girl, aged 13, the aphonia coming on four or five weeks after the illness, the ataxy, the anosmia, and the hypoæsthesia of the whole body, make it practically certain, as pointed out by Determann [33], that hysteria was superadded to the organic condition. In Bosc's case of fatal hemiplegia there were also some symptoms indicative of the presence of hysteria. Thus when the foot was pricked there occurred intense cephalalgia and no sensation localised anywhere else; a prick or heat applied to the left arm caused the same severe head pain, which was otherwise absent, together with pain in the right arm; these symptoms are known to be characteristic of hysteria. Nothing but slight hypoæsthesia was present on the hemiplegic side at first, and the allochiric condition came on only later. Further, the patient had for a year been confined for "melancholia."

To sum up, out of these twenty-nine undoubted cases of true allochiria, in twenty-six there is no reason to suppose that any other nervous condition besides hysteria was present; in the other three, cases of tabes, hemiplegia, and diphtheritic paralysis, there was reason to suppose that hysteria was present in addition to the organic affection. The overwhelming importance of hysteria in this connection is thus obvious, a conclusion that can be supported by evidence of quite another nature.

III.—Description of True and False Allochiria.

I hope later to publish a detailed account of some cases that I have studied, together with a discussion of the pathogenesis of the different conditions. In the present paper I propose to offer only a descriptive account of the clinical features of the conditions, based on the observations made in connexion with these cases.

(1) *Separation of Dyschiria from Alloæsthesia.*

It is the main thesis of this paper to maintain that when a patient makes a mistake in naming the side of a

cutaneous stimulus that mistake may arise in two fundamentally different ways and from two fundamentally different conditions. These two different kinds of mistakes have in the past been confused under the one name of allochiria, and, further, an essentially identical pathogenesis has incorrectly been thought to underlie both. Now only one of the two conditions corresponds at all closely with the definition originally given by Obersteiner, so that the other condition, called above false allochiria, has no claim at all to the designation of allochiria ; this in spite of the fact that Obersteiner, who, like all subsequent writers, did not recognise the distinction between the two classes, gave instances of both conditions in the cases he described in his original article. The one condition, false allochiria, is nothing more or less than alloæsthesia. The other condition comprises three sub-varieties. I have proposed [79] to call this group dyschiria,[1] reserving the term allochiria for one of the varieties, for reasons which will presently be explained. The only substantial advance in our knowledge of the subject since Obersteiner's writings was made by Pierre Janet [70]. Janet observed in a case of hysteria that the allochiric manifestations could be clearly divided into three stages which, described shortly, were as follows : In the first stage the patient did not know the side of the stimulus, in the second she referred it to the opposite side, and in the third to both sides. These stages will be more fully described presently. Janet, however, probably because he recognised only some of the manifestations characteristic of the condition, did not appreciate that the condition was essentially different from the error in alloæsthesia, and hence concluded, like all other writers, that it might occur in a number of diseases, including tabes, hysteria, &c. [71].

[1] This word emphasises the fact that the condition is primarily a disturbance of the sense of right- or left-handedness. Certain writers, such as Gay [48] and Longuet [92], have objected to the propriety of the term allochiria, first employed by Obersteiner [113], on the ground that the symptom in question is more often met with in the feet than in the hands. The use of the root χειρ may, however, be defended for the first-mentioned reason. Its appropriateness will naturally appeal most to those nations, such as the English, and to a less extent the Germans and Italians, who habitually use the word "hand" to denote the right or left side, and must be less obvious to those, such as the French, Dutch and Spanish, who less frequently use it for this purpose.

Amongst other decisive reasons for separating these two conditions I might at the outset mention the following. If we refer to Obersteiner's definition given above we see that the chief point he lays stress on, and which constitutes the essence of his discovery, is the fact that certain patients are in doubt or error about the side of a given cutaneous stimulus *while cutaneous sensibility and the power of localisation are otherwise retained;* in other words, that there exists a specific defect relating to the determination of the side of a stimulus. As will be explained later, it is more accurate to substitute the word "ignorance" for "doubt" in this definition, but otherwise it remains to-day as the most accurate, though of course incomplete, description of a certain peculiar condition. Now this condition is dyschiria and not alloæsthesia; the essential point of the definition applies exactly to the former and in no sense to the latter. In dyschiria the failure to determine the correct side is a specific failure and is quite independent of any other failure as regards perception of the stimulus. In other words, the patient may recognise every single feature concerning a given stimulus—its precise nature, position &c.—except the one point of its side. The evidence goes to show that there is present a mental defect of the specific feeling of "sidedness," of what may be called the chirognostic sense. On the contrary the mistake in alloæsthesia is essentially due to incomplete perception of the stimulus and is accompanied by many other failures in perception, such as defective localisation. Janet, on the other hand, considered that in both the hysterical and organic cases (here called dyschiria and alloæsthesia respectively) the defect was dependent on hypoæsthesia, and that the only difference in the two cases lay in the fact that in the former the hypoæsthesia is of psychical, in the latter of physical origin [72]. My observations have convinced me that no hypoæsthesia, even in the faintest degree, is necessary for the production of dyschiria, and that the hypoæsthesia which is often present is quite fortuitous and occurs merely because it happens to be a frequent symptom in the affection producing dyschiria.

We may now pass to a consideration of the clinical

features that characterise the conditions in question. Alloæsthesia is such a well-known condition that no special description of it need be given except in so far as the points differentiating it from allochiria are concerned. Separate accounts of the three varieties of dyschiria will first be given and then some features common to all mentioned. There are three manifestations of each variety—sensory, motor and introspective.

(2) *Description of Dyschiria.*

(*a*) *Achiria.*

For the first variety, which is identical with what Janet [76] called "simple allochiria," and which possibly corresponds with the state of confusion that Bosc [11] spoke of as "False Allochiria," I have proposed the term Achiria [80], for its most striking feature is the failure as regards the feeling of "sidedness" or "handedness."

(1) *Sensory.*—A stimulus applied to the affected part arouses no feeling of "sidedness" whatever. It is not accurate to say that the patient is in *doubt* as to which side the stimulus has been applied; he is quite sure that he has no idea on the subject and refuses to make any guess. All he can say is that such and such a stimulus has been applied to such and such a part of the body, but as to which side of the body he has no notion.

Apart from this single fact there is no defect of sensorial acuity, even with the most rigorous testing. The stimulus is perfectly appreciated, its exact nature recognised, and its position correctly localised.

(2) *Motor.*—If the patient is asked to carry out any movement with the limb in question he is unable to do so unless the limb is indicated in some other way than by the use of the words right and left. The reason for this is that he has lost the knowledge of the meaning of these words, either altogether or at all events when they are applied to the limb concerned.

(3) *Introspective.*—The patient has lost the memory for the feeling of the part of the body concerned, and declares that though he knows he has such a part he cannot feel it.

(*b*) *Allochiria.*

The second variety corresponds with what Janet [73] called "complete allochiria" and Bosc [12] "true allochiria." I would suggest that the term "allochiria" be retained solely for this variety, the condition in which stimuli are constantly referred to the corresponding point on the opposite side. This restriction of the term, besides giving us an increase in precision, has the further advantage of agreeing with the sense in which it has been used by every writer other than Obersteiner.[1] As stated above, Obersteiner's definition applies to the group here called dyschiria, while all subsequent writers have selected one feature in his definition and called it allochiria. That feature is the characteristic of the condition here also called allochiria, so that my suggestion is in conformity with the accepted use of the term, though it departs from the way in which Obersteiner defined it. The three manifestations are as follows :—

(1) *Sensory.*—Stimuli applied to the affected part are invariably referred to the corresponding point on the opposite side of the body. This is done with an air of absolute conviction, so that, for instance, a patient showing allochiria on the right side only feels no more certain that a stimulus is on the left side when it is applied on this side than when it really is applied on the right. The point to which they are referred on the opposite side corresponds exactly with the symmetrical point touched, a fact which in itself disposes of the view that allochiria is in any way merely a disturbance of localisation. As will be mentioned later the symptom may be present in connection with some kinds of stimuli and not others ; thus painful stimuli may be referred, but not tactile, but when one given stimulus is referred then all similar ones are also ; it is not a question of some or most, but every one without exception.

(2) *Motor.*—If the patient is asked to carry out a movement on the affected side he does so with the corresponding part of the opposite side, fully under the impression (if his

[1] With the exception of Janet, who uses it in Obersteiner's sense.

eyes are closed) that he has correctly performed the required movement. The term allokinesia has been used—see, for example, Blocq's definition [9]—to indicate this symptom, which has been described in cases of hysteria by Binet [5], Janet [77], Féré [41], and others; Blocq says that it occurs only in this affection. It seems unnecessary to use a separate term to indicate the symptom, unless it be found convenient to add the prefix "motor," as Lapidous [85] did. The use of a separate term has in the past served to create an artificial separation between two aspects of what is essentially the same condition. Allochiria is neither a motor nor a sensory phenomenon, but a psychical phenomenon having both motor and sensory manifestations. An instance of what may result from the non-recognition of this fact and of the importance of suggestive influence is found in the cases recorded, for instance, by Trapieznikow [128], Dumontpallier [38], and Binet and Féré [5], in which stroking one side led to movement of the opposite arm. Here the allochiric patient felt that he was expected to make a movement in response to the stimulus, and moved the arm on which he felt the stimulus, *i.e.*, on the opposite side. I need hardly say that such cases in the past have led to other interpretations than the simple one here offered.

(3) *Introspective.*—The chirognostic sense is altered as follows: In a bilateral case the patient can appreciate a given feeling of "sidedness" only when the opposite limb is moved or stimulated. In a unilateral case he can appreciate the feeling of "sidedness" of an affected part only when he is moving the corresponding part of the opposite side under the impression that he is moving the part in question; if he really moves the affected part, or if this is stimulated, he invariably gets the feeling of "sidedness" of the opposite part.

(*c*) *Synchiria.*

To the third variety Janet [74] has given the appropriate name of synchiria. The manifestations of it are as follows:—

(1) *Sensory.*—A stimulus applied to the affected part evokes two simultaneous sensations, which are referred to

the corresponding points on both sides of the body. Either or neither of these two sensations may seem more distinct than the other, so that three substages of the condition can be distinguished.

(2) *Motor.*—When the patient is asked to carry out a movement on the affected side he does so on both sides though in so doing he gets only the feeling of "sidedness" of the affected part. This symptom reminds one superficially of the synkinesia seen in organic hemiplegia, though, of course, the pathogenesis of the two symptoms is entirely different. It is undesirable that the term synkinesia should be used for two phenomena, one physiological and the other psychological, especially when we have the more descriptive term "motor synchiria" available for the latter; this confusion shows further how unfortunate was the choice of the term allokinesia to designate motor allochiria, for the use of it tends to involve the use of the term synkinesia in an ambiguous sense.

(3) *Introspective.*—The patient is unable, either spontaneously or when cutaneous stimulation is applied, to appreciate the affected feeling of "sidedness" alone apart from the simultaneously appreciated feeling of the corresponding opposite side, though he can appreciate it when he moves both limbs together under the impression that he is moving only the affected one.

We have next to consider some features that refer to all varieties of dyschiria.

(3) *Distribution of Dyschiria.*

Dyschiria may occur in relation to any or every segment of the body. In the cases recorded by Ferrier, Janet, and Gay it was bilateral and general. In some cases, as in those recorded by Dobie, Bosc, Féré, and Magnin, it may be restricted to certain regions of the body, even to only one conjunctiva. In certain cases, such as in Ferrier's, the distribution may vary at different stages of the complaint.

In the same part of the body dyschiria may be manifested in connection with all or with only some of the functions of the part. It may thus concern only motor

functions or only sensory or both. Further, it may be manifested in connection with all varieties of sensibility or only with some; for instance, in Dobie's case of allochiria only painful stimuli were referred to the opposite side. This interesting dissociation may be otherwise expressed by saying that only certain kinds of stimuli can arouse the appropriate feeling of sidedness and not others, an occurrence highly characteristic of the specialised disaggregations of hysteria. As stated above, however, if a dyschiric manifestation is shown in relation to a given kind of stimulus—for instance, a painful one—it is constantly and invariably shown with every repetition of the same kind of stimulus.

(4) *Sensibility in Dyschiria.*

Two facts should be prominently stated in this connection. First, that though defects in sensibility often, perhaps even usually, occur in accompaniment with dyschiria, they bear no relation to this symptom; they vary independently of it, and finally they need not be present at all. With the most careful examination I have found sensory acuity to be perfectly normal in each of the three varieties of dyschiria. Secondly, in all stages of dyschiria there are certain peculiar attributes constantly present in sensation evoked by stimulation of the affected part. I have proposed the name Phrictopathic Sensation [80] to indicate sensation having these special attributes—six in number—and need not describe them further here, especially as I hope soon to publish an analysis of the phenomenon.

(5) *Clinical Course.*

In the twenty-nine hitherto published cases of dyschiria, allochiria was present in all, synchiria in two (Ferrier's and Janet's), and achiria in only one (Janet's.) There is much experimental evidence that goes strongly to show that allochiria is the stable state of the three, and that though allochiria may probably occur without the other two ever being manifest, the reverse is most unlikely. This would, of course, explain why allochiria is the variety that has

been most frequently observed and why attention has, apart from Janet's writings, been confined to this variety. Of the three varieties achiria represents the most advanced stage of psychological disaggregation and synchiria the least. Most of the cases were apparently observed for only a very short period, so that we have little data to decide the question of how long the syndrome may last. That this may be for a considerable period is, however, certain from the fact that in Janet's case allochiria was present for at least twelve years, and in one of mine for probably six years.

(6) *Relation to Consciousness.*

Like so many hysterical defects the dyschiric manifestations are more marked the more clearly conscious the mental process concerned. This may be demonstrated in both the sensory and motor aspects, but I will content myself here with the following illustration taken from the latter. One of my patients with unilateral allochiria could walk perfectly well in the ordinary way when not paying attention to the act, but whenever he became specially conscious of the necessary movements the allochiric disturbance manifested itself. Thus when trying to step on to an omnibus with the affected leg "both legs wanted to move at once," as he put it, and he would frequently topple over.

IV.—Theory of Allochiria.

In this section I propose to give a criticism only of previous hypotheses in the light of my own observations, for a full discussion of the actual psychical pathogenesis of the condition would lead us too far from the clinical purpose of this paper.

The current and most widely accepted explanation of allochiria is usually styled "Hammond's theory," though all the essential points of the hypothesis had previously been described in 1880 by Fischer [47]; it has been well expounded and developed between 1887 and 1900, particularly by Huber [67], Weiss [134], Bosc [15], Mott [103],

Gay [49], and Determann [34]. As given by Hammond [58], this view runs as follows: Starting from the assumption that "it is quite certain that there is an almost complete decussation of the sensory fibres within the grey matter" (page 36), he concludes that "with a lesion of one posterior horn the sensation would be directed through the grey commissural fibres to the other posterior horn and would reach the cortical centre in the corresponding hemisphere" (page 37), the sensation being then referred by this hemisphere to the wrong side of the body. If we do not enquire too closely into the nature of the process by which the "sensation" is deflected through "grey commissural fibres," this hypothesis presents on the surface an attractive simplicity, and most later writers have found it irresistible, failing thus to appreciate the difference between the simple and the true. Hammond goes on to say that if another unilateral lesion supervened at a different level from the first, the sensation that was previously deflected to the wrong hemisphere was now rediverted by meeting with another obstacle, and so arrived at its proper hemisphere. To quote further: "Such lesions explain those cases in which absolute anæsthesia on one side of the body exists, with sensation on the other side for impressions coming from both sides." It is difficult to interpret this confused passage and to see how there can be sensation on one side for impressions coming from the other if there exists absolute anæsthesia on this other. It is to be supposed that what the writer meant was that allochiria occurs equally with unilateral and bilateral lesions—so long as the latter are asymmetrical—though it is on different sides in the two cases. Hammond also applied his hypothesis with equal confidence to the explanation of the Brown-Séquard hemisection phenomena. He says: "This circumstance, which has not been hitherto explained, is, I think, satisfactorily accounted for by the theory I have proposed. For the parts below, corresponding to the cut half of the cord—for example, the right—not only remain in undisturbed relation with their proper cortical centre in the left hemisphere, but this latter receives also the sensory impressions coming from the left side.

There will therefore be increased sensibility in the left side. Numerous facts in morbid anatomy and pathology could readily be brought forward in support of this view." None of these facts are quoted, however, nor does Hammond make it clear in what way a greater inflow of impressions is synonymous with cutaneous hyperæsthesia.

In spite of the obvious defects of the above hypothesis, it has been considered an adequate explanation of allochiria by the majority of writers on the subject. The hypothesis has been accepted not only by the authorities cited above, but also by Obersteiner [112], Brown-Séquard [23], Gellé [52], Morselli [101] and by von Leyden and Goldscheider, writing in the current edition of Nothnagel's "Specielle Pathologie und Therapie" [89]. Some of these authors have found it quite illuminating; Gellé calls Hammond's ideas and diagrams "très demonstratifs"; Brown-Séquard: "Je ne crois pas qu'il y ait dans les faits d'allochirie rien de difficile à expliquer aujourd'hui que l'on sait que les deux moitiés de la moelle épinière, comme les deux moitiés de l'encéphale, ont de très nombreuses communications et que chacune de ces moitiés peut remplir le rôle de l'autre. On comprend donc aisement comment les impressions sensitives venues d'un côté du corps peuvent être perçues comme si elles provenaient du côté opposé." Weiss enthusiastically says that "diese von Hammond für einen speciellen Fall aufgestellte mit den physiologischen Thatsachen sich vollkommen deckende Theorie kann meiner Meinung nach ungezwungen für alle Fälle Geltung haben."

Now quite apart from any psychological considerations, there are insuperable objections to Hammond's hypothesis, even if this be viewed from his own standpoint. The whole superstructure of his speculations rests less upon a basis of neurological fact than upon the insecure foundations of an argument by analogy. The mechanism of the paths in the spinal cord is assumed to be comparable with that of a railway, as is indicated by the phraseology employed, blocks, signals, tracks, switches, shunts, and junctions being pictured in the most realistic manner. Then by way of finding out what happens in the spinal cord under certain circumstances,

the results of analogous emergencies are studied on the railway, and the information thus gained is applied in the most literal manner to the spinal cord. Hence the conclusion that a message, finding a block on its normal route, changes to another line which unfortunately brings it to the wrong destination. Hence also the irritability or hyperæsthesia that ensues from the congestion of traffic due to the overworking of the second line. If by a happy chance another block leads the message to take again the original line it now arrives at its proper destination, a little delay, however, being noticeable.

A word may be said on the *a priori* improbability of this conception of the functions of the spinal cord. The cord is, phylogenetically speaking, one of the most ancient structures in the body, having been gradually evolved during many millions of years. As might have been expected from this consideration, all the evidence we have as to its physiology and pathology points to its functioning being of the most fixed and stereotyped nature. The very fact of its relative minuteness in man, as contrasted with lower animals, compels us to regard it as a tightly packed bundle of constituents, each having a highly differentiated and predetermined function. The conception of it as a plastic and relatively homogeneous whole, the different parts of which are prepared at a moment's notice to take over all the functions of other parts, thus conflicts violently with all we know of its history and structure.

Even, however, if we grant for a moment the likelihood of the proposition, we still find that it is out of accord with some commonly known facts concerning allochiria. Thus Longuet [93], after rather ironically remarking in connection with Hammond's explanation of the Brown-Séquard phenomenon, "Ce schéma donne également la clé de ce fait de médecine expèrimentale qui a tant intrigué les physiologistes depuis Galen," points out that the hyperæsthesia of this phenomenon should always be accompanied by allochiria, which is far from being the rule. More striking than its not being the rule, as Longuet cautiously remarks, is the fact that no case has ever been published in which it has

been observed, for the allochiric symptom mentioned by Brown-Séquard and Spearman in their cases is evidently not true allochiria, since the sensations were localised at various distant points, *some* of which happened to be on the opposite side. No cases of this nature have been more carefully investigated than the eight recently published by Head and Thompson [59], and in none of these was transference of sensation observed. Even the most carefully performed experiments on the lower animals have never resulted in the production of allochiria. It is true that Mott in his well-known hemisection experiments on monkeys [104] observed transference of sensation, which he explained by the crossing of impulses to the opposite side, but it is clear that we have here again to do with alloæsthesia and not allochiria, for Mott says [105] that there was great disturbance of localisation in the paralysed limb, and evidently the whole sensibility of the limb was greatly impaired. So far as I know even this false allochiria has not been observed in other extensive series of hemisection experiments, such as those made by Aldren Turner [129].

We are left, therefore, with the difficulty of understanding why allochiria, if it is due to the presence of a unilateral lesion, is rarely if ever seen when such a lesion occurs; Hammond insisted, on the contrary, that such a lesion must uniformly bring about allochiria. A further difficulty is how to explain by this hypothesis the short duration of the symptom which is a manifest feature in most of the recorded cases. Huber, who fully accepts Hammond's hypothesis and calls it "durchaus wahrscheinlich," explains the recovery in his case, one of disseminate sclerosis, by assuming the appearance of a new lesion on the opposite side from that of the first, the fresh block rediverting the impulse towards its original destination [67]. He expresses no surprise, however, at the non-appearance of allochiria on the opposite side, which surely might have been expected, unless one supposes that the second block beneficently limited its diverting influence to those impulses that were travelling on the wrong line. Weiss, who is in general a warm adherent of Hammond's diverting hypothesis, rejects Huber's application

of it to the recovering cases on the ground that it is too complicated [131]. He attributes the disappearance of the symptom to retrogression of the lesion, so that the track is again cleared. This is, of course, a large assumption to make in the case of tabes and certain other cord diseases.

The incapacity of the hypothesis to assimilate further aspects of the problem was most strikingly shown when observations began to multiply in which the spinal cord was normal throughout. Huber, in 1888, instituted the division of cases into spinal and non-spinal [67], but attempted no explanation of the latter group. Weiss, in 1891, rejected this division, as also that of Obersteiner, into spinal and hysterical, and considered that all the cases were equally explicable on Hammond's hypothesis. The only difference he saw between the hysterical and the organic cases was that in the former case the block in the posterior columns was of a functional nature [134]. He does not attempt to clarify this vague notion of a local obstruction of a functional nature, which is difficult seriously to discuss. Determann has severely criticised its glaring inconsistency with all modern knowledge of hysteria [35]. In 1892 Bosc [16], by the publication of his fatal case of hemiplegia, showed conclusively that allochiria could occur apart from cord affections, and divided the cases into spinal and cerebral, including hysteria under the latter term. He considered that all the organic cases were explicable on Hammond's hypothesis, and accounted for the hysterical cases by postulating "an altered dynamic state of the cerebral hemisphere concerned, of such a nature that the impulses reaching it crossed through the corpus callosum to the opposite hemisphere, which therefore became endowed with the dynamic state of both hemispheres" [17]. It cannot be maintained that this conception greatly furthers our comprehension of the condition. He expounded the railroad hypothesis at great length and introduced a new terminology, based on the modern tendency to electrification, in his remark that "the grey matter plays the *rôle* of a commutator" [15]. Determann, in his monograph in 1900 [35], took up much the same standpoint as Bosc.

Ever since Bosc's writings all writers have agreed that Hammond's explanation was of limited applicability only, and that there was definitely a group of cases for which some other explanation would have to be sought. Morselli [101] still further limited the range over which it could be applied by rejecting it in connection with all the cerebral cases, though he still accepted it as explaining the spinal cases; he offered no suggestion for the former group. It would be very curious if a peculiar phenomenon like allochiria could be produced in two quite different ways, and Grainger Stewart in 1894 [125] voiced a very obvious reflection when he said that he "doubts whether the blocking hypothesis proves very satisfying to anyone." He further remarked that the results of his study of a case of alloæsthesia "lend some support to the opinion that in allochiria the fault is not so much in the conducting fibres as in a morbid action of the centres."

So much for the physiological aspects of the subject. On this side speculation has come to a full stop, and writers who take the physiological standpoint can be divided into those who accept without analysis a doctrine that is incompatible with the facts, and those who despondently admit their failure to understand any of the mechanism of the phenomenon. It was only when the subject was viewed from a psychological standpoint that any light at all was thrown on it. The most important contributions from this standpoint are those of Pierre Janet, his first in 1890 [69] being amplified in 1893 [78] and 1898 [70]. Janet's views have not received the attention they deserve. A short summary of them is given by Binswanger [7] and Lapidous [87], who, however, offer no criticism or further suggestions; both refer to them only in connection with hysteria, and Lapidous expressly commits herself to the belief in three groups of cases—hysterical, cerebral, and spinal, accepting Hammond's explanation of the latter two [88]. Trapieznikow [128] agrees with Janet that an anatomical origin of the phenomenon cannot be accepted, and that it must be considered as a perception defect. He hints that the psychic explanation is applicable to all cases and rejects the division

into spinal and cerebral. He contributes nothing to the elucidation of the mechanism of the phenomenon.

Important contributions to the psychology of the problem have been made by Henry Head [60] and Spearman [124]; Victor Henri has also referred to the subject from this standpoint [62]. Although these last three writers use the term allochiria it is clear that the condition they were discussing was that of alloæsthesia, in which the symptom we have here called false allochiria may appear. Their contributions are therefore of value only so far as this condition is concerned. From the enormous mass of work done on the complex subject of localisation by these and other authorities, an adequate explanation of the nature of alloæsthesia seems definitely to emerge. This is perhaps most precisely voiced in Spearman's statement that the main factor in the production of the condition is a defect in the "articular" excitations; the "segmental" excitations supply very imperfect material for the accurate determination of the position and side of the stimulus, so that when they alone are relied on, and especially when they too are deficient on account of cutaneous hypoæsthesia, the failure becomes manifest. This defect may occur in hysteria, but is more usually due to a gross lesion in the projection system of afferent fibres, and it is interesting to note that the disease in which the greatest defect in "articular" excitations occurs, namely tabes, is the disease in which the symptom of false allochiria, due to alloæsthesia, has been most frequently described.

It would be impossible adequately to discuss Janet's sychological considerations here, and I will add nothing to the remarks offered above (*see* Introduction to Section III.), which sufficiently indicate the difference of my standpoint from his. My observations point to the view that dyschiria is due to a psychical affection of the feeling of "sidedness," and that the mechanism whereby the different varieties of the syndrome are produced is also psychical in nature; the actual problems of pathogenesis I hope to discuss in a future paper.

V.—Diagnosis of Dyschiria.

It will be convenient to consider separately the sensory and motor aspects of this problem. The discussion of them may be prefaced by the general remark that if the facts of dyschiria, as described above, are borne in mind, it is practically impossible to make any mistake in the diagnosis of the condition. In the absence of this knowledge, however, there are a number of ways in which the various symptoms may be overlooked or misinterpreted, and as the condition frequently goes unrecognised it is perhaps worth while to mention the possible fallacies of observation in some detail, elementary as they may seem. Dyschiria is interesting in this respect as being one of the rare examples in medicine in which erroneous diagnosis is due rather to ignorance of a few simple facts than to any failure in judgment.

(*a*) *Sensory.*

In the first place it is evident that the sensory manifestations will probably be altogether overlooked if, when testing a patient's localising capacity, the observer neglects to enquire expressly as to the side to which the sensation is referred. This omission is especially likely to be made when, as in dyschiria, the patient shows no defect in sensorial acuity and localises the stimulus with exactitude and certainty. Under such circumstances, even if the patient mentions the wrong side, this may be attributed to a slip of the tongue, and the matter not pursued any further.

I have formulated elsewhere [80] seven precise differentiating features between allochiria and alloæsthesia, so that they need not be recounted here.

All the three subvarieties of synchiria may give rise to special fallacies. In the first and most advanced of these the contralateral sensation is felt to be more distinct than the other, and if the patient mentions only the more distinct one the condition is liable to be mistaken for allochiria.

A patient may say that he cannot tell on which side certain stimuli are applied. If this observation is correct it is pathognomonic of the existence of achiria, provided that

sensibility is intact. As, however, this condition has never been described in any neurological journal or text-book, the observation has a considerable chance of being misinterpreted. There is one fallacy in connection with it, namely, that a patient in the second stage of synchiria is liable to make the same answer, if he thinks that only one stimulus has been applied, for he feels two sensations equally distinctly and cannot decide between them; if this fallacy is not avoided a serious prognostic error may be committed.

In the third subvariety of synchiria the homolateral sensation is the more distinct, and if the patient mentions only this one the state of affairs may be thought to be normal.

The only way of avoiding with certainty the above fallacies in connection with synchiria is to put the leading question to the patient as to whether he feels the stimulus on both sides or on only one.

Lastly, it should be stated that just before synchiria is replaced by the normal state there is a period when the patient feels only a homolateral sensation. If sensibility is being tested at this time, unless the phictopathic features of the sensation are noted the condition is almost certain to be overlooked. As this period may be of only momentary duration it is easy to see how the clue to the various other phenomena present may thus be completely missed.

Finally, it must be remembered that the dyschiric manifestations may be confined to certain regions of the body only, and may relate only to certain kinds of stimuli.

(*b*) *Motor.*

On the motor side the possible errors in observation are both more important and less obvious. The patient's symptoms may be described in a very misleading way. Unless the motor manifestations are carefully analysed they may, particularly in achiria, readily be interpreted as clumsiness and weakness. Tests for motor incoördination elicit fumbling movements badly adapted to the end in view, and when the patient's strength is tested directly—for instance, in the performance of such a simple act as hand-gripping—

the conscious effort produces such disproportionately slight results that the presence of marked paresis is readily assumed.

The functional basis of the motor manifestations will probably be recognised if their distribution is general and bilateral, but it is less likely to be when the distribution is monoplegic or hemiplegic. In the latter instance the resemblance to incomplete hemiplegia is very striking, as may be better realised by shortly considering the notes of one of my cases made when the patient came under observation.

In the achiric stage this patient complained of weakness and awkwardness of the right side. Examination apparently confirmed the truth of this statement and, as occurs in all varieties of hemiplegia, the defect was most marked for acts consciously performed; in fact it was present only in relation to such acts. The immediate reason of the patient's coming to hospital was his alarm at the fact that in an automatic state he had felled a man to the ground the week before. The same hand could with a dynamometer register only 5 lb. The gait was that of a hysterical hemiplegia, the right leg being dragged in the inert manner so long ago described by Todd. All the signs of supranuclear facial paralysis were present in their entirety, including the shallowing of the naso-labial furrow, non-movement of the side volitionally, with retention of emotional movements, Revilliod's orbicularis sign, &c. The deep reflexes were active, a marked tendency to ankle-clonus was present and sharp extension of all the toes followed plantar stimulation, the resemblance to Babinski's sign being, however, incomplete. Under these circumstances it is comprehensible how more than one neurologist had previously made in this case the diagnosis of hemiplegia of organic origin. Even if the case had been called one of hysterical hemiplegia the error in diagnosis would have been serious, for re-educative measures might thereby have been directed to the motor aspect instead of to what is much more nearly a sensory aspect.

A symptom is frequently met with in all the dyschiric

stages that may readily be mistaken for aboulia, a mistake that would also direct treatment on quite erroneous lines. When the patient is asked to perform a simple act no movement may result in spite of his efforts; this can, however, be at once told from aboulia in that the patient, unless he is watching the limb, is under the full impression that he has carried out the act successfully and distinctly feels the limb move, although in fact it remains at rest. Consequently there is none of the recognition of failure and feeling of powerlessness that characterises an aboulic manifestation. Further, there are objective evidences that effort is actually being put forth, so that the failure in performance is not due, as in aboulia, to defective effort.

In synchiria the bilateral movements, sometimes called synkinesia, bear a decided resemblance to the synkinesic phenomena of organic hemiplegia. Especially is this so in the third variety of synchiria, when the contralateral movement may be less in degree than the homolateral. The two phenomena may, however, be easily distinguished by the other characteristics of each.

Certain respects in which motor allochiria may be misinterpreted were referred to when we discussed this condition, but the errors there mentioned are not likely to be committed at the present day, as the importance of suggestional influence is more widely recognised. In this way, for instance, may be explained Dumontpallier's case [39], in which stimulating one limb caused contralateral contraction of the other.

VI.—Diagnostic Value of Dyschiria.

In the literature the indications in this direction are but scanty, as might have been expected. Musser's opinion [106] has been quoted above, to the effect that allochiria, when general, should be regarded as a stigma of hysteria, and when local usually occurs with organic disease of the spinal cord. Determann [36] cautiously remarks that when *both* sides of the body are allochiric for *all* sensations one should think of hysteria. Otherwise writers, from Obersteiner [112] to Janet [75], are almost unani-

mous that allochiria is a symptom that may occur in a multitude of affections and is practically valueless in diagnosis.

The evidence, however, is decisive in favour of the view that dyschiria, including allochiria, always represents a psychical affection of a disaggregative nature, and further, there is considerable evidence relating to the "equivalents" of the syndrome which strongly indicates that the disaggregation is of the kind typical of hysteria, and not, for instance, of the less massive kind that I have suggested is present in psychasthenia [82]. It may therefore be concluded that the occurrence of any form of dyschiria should be regarded as a positive indication of the presence of hysteria. I have related elsewhere [81] some of the arguments that may be advanced in favour of this view, and so need not repeat them here.

The clinical importance of the phenomenon is by no means limited, however, to the mere recognition of the presence of hysteria. Observation of its course and variety may be of great service in the matter of prognosis. Naturally the intensity of the dyschiric defect, as shown by the stage present, has a direct value in this connection.

Recognition of the dyschiric process may throw light upon a number of symptoms that would otherwise be misinterpreted as paresis, aboulia, defective sensibility, &c. The great value of this is that it enables a correct analysis to be made of the precise defects present, and serves as a guide towards the original focus of the whole affection, thus proving an important step in that exact psychological diagnosis that is an essential preliminary to the scientific treatment of hysteria. On the therapeutic side the progressive recovery of the functions in question by the aid of careful training may be utilised as a starting point for the general process of intelligent re-education that is the basis of all rational treatment of the affection.

VII.—Summary of Conclusions.

(1) Under the name of allochiria two fundamentally different conditions have hitherto been confused. A patient's

mistake in determining the side of a stimulus may be—(i.) part of a general defect in localisation—alloæsthesia, or (ii.) a specific defect independent of any error in localisation—dyschiria. The name allochiria has further been incorrectly applied—as in the terms electromotor and reflex allochiria—to symptoms which are in no way related to either of these conditions.

(2) Dyschiria may be defined as a state in which there is constantly either ignorance or error in the patient's mind as to the side of given stimuli, quite independent of any defect in sensorial acuity or in the power of localisation. This corresponds closely with the definition of allochiria given by Obersteiner, though he did not distinguish the condition from alloæsthesia.

(3) There are three stages of dyschiria: achiria in which the patient has no knowledge as to the side of the stimulus, allochiria in which he refers the stimulus to the corresponding point on the opposite side, and synchiria in which he refers it to both sides; there are three subvarieties of the latter.

(4) All writers subsequent to Obersteiner have abstracted one feature from his definition—namely, the reference of the stimulus to the opposite side—and have used it to define allochiria. It is suggested that the term allochiria be always used in this its current sense, with the important proviso, however, insisted on by Obersteiner, that the symptom is independent of any defect in sensorial acuity or in the power of localisation. The significance of this proviso has been entirely overlooked hitherto, and even Obersteiner did not recognise that a direct corollary of it is the separation of the alloæsthesic error from the allochiric error.

There are seven precise clinical features that enable a differential diagnosis between alloæsthesia and allochiria to be made with certainty.

(5) Alloæsthesia, including false allochiria, is adequately explained by the Head-Spearman hypothesis that it is due to a defect in afferent excitations, particularly those of the "articular" type. It occurs in both organic and functional disease, perhaps most often in tabes.

(6) Dyschiric manifestations may be general in distribution or may relate only to certain segments of the body. There are characteristic introspective, motor, and sensory manifestations of each member of the group. The last mentioned may occur in connection with all varieties of stimuli or with only some. The motor allochiria has been unnecessarily termed "allokinesia." Sensation resulting from stimulation of a dyschiric part has six peculiar attributes, which I have grouped under the designation phrictopathic.

(7) There have been three explanations of allochiria hitherto offered. The Fischer-Hammond hypothesis is throughout contradicted by the facts and should be entirely discarded. The Head-Spearman hypothesis refers to alloæsthesia only and had no relation to allochiria. The Head-Janet hypothesis is not borne out by the observations on which this paper is based, which seem to demonstrate that allochiria is independent of any defect in sensorial acuity.

(8) Dyschiria is due to psychical disaggregation and is distinctive of the form of disaggregation characteristic of hysteria. It is primarily an affection of the feeling of "sidedness" (the chirognostic sense).

(9) Of the three stages of dyschiria achiria represents the most severe grade of disaggregation and synchiria the least. These two are essentially transitional forms. Allochiria, on the other hand, is a stable condition which may be present for years.

(10) A number of fallacies in diagnosis are here indicated and especial attention drawn to the close resemblance between unilateral achiria and hemiplegia, particularly hysterical hemiplegia.

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A CASE OF CYST WITHIN THE SPINAL CANAL.

BY WILLIAM C. KRAUSS, M.D.

Of Buffalo, N.Y., U.S.A.

JAMES P., male; occupation, carpenter; age 46; born in the United States; married.

Family History.—Father and mother are both living and healthy. One brother has a history of some spinal trouble, nature not known. One sister died at the age of 34 of so-called "paralysis."

Early History.—When very young he had an attack of peritonitis. At the age of 7 he suffered slightly from sunstroke. When 25 years old he jumped from the rear of a wagon and injured his spine, at about the place where the present lesion is located. This injury evidently was of little consequence, as he recovered quickly and has been an unusually healthy man ever since, up to the time of the beginning of the present trouble. He has always lived in the country, and has been a rugged, robust, out-of-door labourer; no history of syphilis or tuberculosis was obtainable.

Present Illness.—About November 20, 1903, he began to experience sharp, shooting pains in the upper lumbar region. These pains were intensified by rotating or flexing the body, or by raising or using the arms. In a short time the pain extended until it encompassed the body, passing along the lower border of the ribs on the left side; later on the right side was similarly affected.

About January 1, 1905, he began to notice a tingling in the feet, which increased in severity until they were quite numb, and he was no longer sure of his footing; weakness of the legs appeared, and progressed so rapidly that by the end of January, 1905, he had lost all use in them.

On January 5, 1905, he was brought to my office by his family physician, and the following notes were made:—

A well-built, well-nourished man, weighing about 150 lbs., and 5 ft. 10 in. in height. His gait attracts attention at once, being paretic and spastic. He rises from the chair with difficulty and

complains of pain in the back on rising or sitting in the chair. Muscular power of the legs is considerably diminished. The muscles of his legs react fairly well to stimulation with the faradic current, but the reaction is evidently diminished. Ankle-clonus is present on both sides, together with Babinski's plantar reflex. The spine offers nothing noteworthy on inspection, but

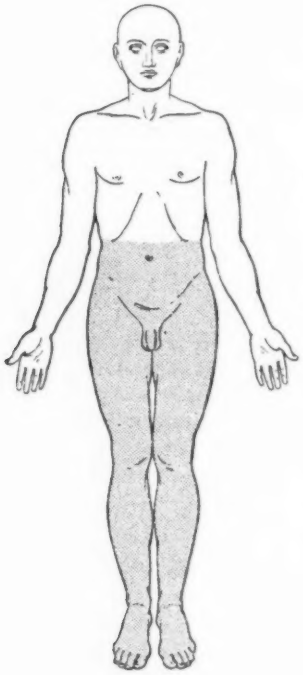

FIG. 1.

To show the Extent of the Diminution of Sensation.

over the fourth and fifth spinous processes of the vertebræ, deep pressure causes considerable pain and flinching. There is nothing abnormal on palpation, no swelling, and no curvature. The sensation is diminished, ventrally corresponding to a line two inches above the umbilicus, while on the dorsum the line of demarcation is inconstant. The bowels are very constipated, necessitating the most active cathartics, and the bladder is paretic, catheterisation

being performed every four or five hours. The appetite is good. Sleep after the back is once in an easy position is satisfactory. Heart and lungs offer nothing abnormal. Temperature and pulse are normal, and urine shows some leucocytes, evidence of a mild degree of cystitis.

A positive diagnosis was not made at the time, but I suggested the strong probability of a spinal-cord tumour as the cause of the paraplegia and severe back pains.

The patient returned to his home in Alabama, N.Y., thirty miles distant from Buffalo, and was put under symptomatic treatment. As his condition did not improve I was called to see him at his home at Alabama, and found his condition as follows:—

He was no longer able to stand on his feet, and was lying on the couch when I entered. There was now a complete paralysis of both legs, which were œdematous, pitting on pressure, mottled blue in colour and cold to the touch. The patellar reflexes were very much exaggerated; patellar and ankle-clonus were present on both sides, together with Babinski's plantar reflex. Bladder and rectum were paretic. Examining the spine, a sensitive area was found over the third, fourth and fifth thoracic vertebræ, and this area was swollen and bulged perceptibly. The level of the diminished sensibility corresponded to a line two inches above the umbilicus ventrally; while on the dorsum, a sharp line between areas of normal and impaired sensation could not be definitely determined. The abdominal reflex could not be elicited. The patient complained of twitching in the legs, and when lying on his back the legs would draw up spasmodically.

A diagnosis of spinal-cord tumour was definitely made, and the patient urged to enter the Buffalo General Hospital at once for an operation.

From time to time I received word from the family physician of his condition, and on February 6 was given a small bottle containing a sero-sanguineous fluid, which had been withdrawn with a syringe from the swelling in the back. It appears that the pain became so intense and the tumefaction so pronounced and sensitive, that the family physician decided to insert a needle and draw off some of the fluid. Prompt relief was afforded by the withdrawal, and the patient again was shown the benefit which he might obtain if a radical operation were undertaken. Consent to an operation was obtained on condition that it be performed at his home in Alabama. This was acceded to, and Dr. Roswell Park was called at once into the case. During the week preceding the operation, a slight increase of temperature was noted and the

subjective pains were considerably diminished in severity. No bed-sores had formed, although patient had been bed-ridden for nearly three weeks. Dr. Park's report is as follows:—

"Symptoms date back to about twelve weeks ago. They began with a rather sudden onset of pain and weakness of muscles of the leg. Pain was referred to the lower abdomen. His condition has been aggravated, and for the past two weeks he has been bed-ridden and paraplegic. The area of lost sensation points to involvement of the cord at about the height of the fourth or fifth dorsal vertebræ; it varies a little almost from day to day. He has had no girdle pains, and his first symptoms were confined rather to the left leg, but now they are equally distributed, and there is no essential difference between the two sides of the trunk or the legs. The bladder and bowels are paralysed. Appetite fair; general condition fair. Of late he has been running evening temperature of about 100° F. In the mid-dorsal region there seems to be a little projection, and at about the level of the third dorsal spine there appears to be a collection of fluid, which a week ago was explored with a hypodermic needle and some bloody fluid, apparently serum mixed with blood, withdrawn. At the point of the puncture there now seems to be a little local infection.

"Symptoms pointed to pressure upon the cord. Dr. Krauss had suggested cysticercus. At all events, patient had been failing of late, and Dr. Krauss urged operation.

"February 13, 1905, at patient's home, I made an incision extending from the height of the third dorsal spine down to the sixth. Not until separating the muscles and tissue from the spines and arches, did I come down upon a cavity apparently between the fourth and fifth spinous processes, into which the finger could be easily passed. It was then apparent that there was a considerable cavity excavated at the expense of the vertebræ, especially the posterior arches, which contained the fluid originally found, and which was now mixed with blood. This cavity was not lined with membrane, but everywhere eroded, and rough bone could be felt. It was therefore not a cyst which could be extirpated. At its base the cord could be felt within the dura lying in the canal and apparently not affected, save by pressure from without. A small amount of granular material was removed with the spoon and saved for microscopic examination. The cavity bled freely, but not excessively. Neither above nor below it could I make out any point at which pressure seemed to have caused constriction of the spinal canal.

"The exact nature of the lesion was not disclosed by the operation, but it seemed evident that there was a cavity with eroded walls connecting with the spinal canal, containing mostly fluid, with a little semi-solid tissue material.

"I inserted a bundle of catgut fibres for drainage and otherwise completely closed the wound, both by buried and superficial sutures."

Following the operation, the patient progressed favourably. An acute cystitis set in on February 17, which was, however, quickly relieved. Ten days after the operation he was sitting up. The sensation began to return on the third day, and has steadily increased. The wound healed by first intention; temperature and pulse remained normal, and on February 27 the special nurse was discharged by the family. Catheterisation was still necessary up to the time of the nurse's departure, and the œdema of the legs still continued, especially of the left leg.

On May 25, 1906, I made a special trip, and found the patient's condition as follows:—

He was found lying in bed reading a newspaper. He had been around in a wheel chair, going to and from the village stores. He said while sitting up he felt as if there were more life in the buttocks, as if he could almost move about. He has not had any pain since the operation, nor have bed sores, or any other trophic disturbances, appeared. He has regained complete control over the sphincter ani, but is obliged to be catheterised daily.

Examination of the back shows the scar to be in excellent condition. The thighs and legs are not atrophied. The patellar reflexes are exaggerated, ankle clonus and Babinski's reflex are present on both sides. Muscle contractions are frequent, and sometimes almost painful.

Sensation.—There is found a narrow zone of hyperæsthesia at the tenth thoracic segment, and anæsthesia for pain, pressure and temperature.

Motion.—He has considerable strength in the hips, and can move the thighs slightly. He can exert no power over the legs, feet or toes whatever. The feet are inclined to cross, due to involuntary contraction.

His general health is good, and he seems contented with the result of the operation.

There has been no change in the extent or degree of the sensory disturbances, some slight recovery of motor paralysis, some control over the sphincters, and the abolition of all subjective symptoms.

Although the recovery in this case is far from being complete, and certainly not satisfactory, yet it must be admitted that the operation was indicated, that it served to check the further progress of the disease, and converted a wretched, pain-racked, rapidly-failing patient into a comfortable, hopeful, but helpless, invalid.

Frequency of Spinal Cord Cysts.

Schlesinger, in his monograph on spinal cord tumours, analysed 400 cases, without reference to operation, and found eight cases of cysticercus, forty-four of echinococcus, and two of other cysts—in all fifty-four cases of spinal cord cysts, or about 13 per cent. of all tumours reported. Thirty-nine of the echinococcus cysts were extradural, and of these twenty were multiple, twelve undecided, and seven were solitary. Of the intradural echinococci four were multiple and one solitary, making a total of twenty-four multiple and eight solitary cysts, and twelve cases undecided.

The seven of the eight cases of cysticercus cysts, on the contrary, were intradural, and of these four were solitary and three multiple. One cysticercus was a multiple extradural cyst. Of the two unclassified cysts, one was a solitary intradural, and the other was multiple and extradural.

Up to 1906 I have been able to collect 105 cases of spinal cord tumour which came to operation, and of these only eleven cases of cysts were noted, or not quite 10 per cent. These cases in order of time of operation will be found in the summary.

Wilms, in reporting the Trendelenburg case (No. 5), cites twenty cases of echinococcus disease of the spinal cord, only three of which came to operation, cases 3, 6 and 8 of my list.

Of the eleven cases which I have found, seven were echinococcus cysts, and four were non-parasitic, cases of Spiller (No. 9), Schmidt (No. 10), my own case (No. 11), and an unique case of dermoid cyst (No. 7), reported to the Clinical Society of London, March 23, 1900, by Hale White. Schmidt believed that his case was the only case on record, but it had been preceded by Spiller's case.

Whether the non-parasitic cysts were of inflammatory

origin or whether they were myelocysts, or pialcysts, is difficult to determine. It is rather of a coincidence that three such cases should be reported within a period of less than three years. Nine of the eleven cases were of extradural origin; two, the cases of Spiller and Schmidt, were intradural.

Cysts of themselves offer no special symptoms which can be differentiated from the ordinary compression symptoms of cord tumours; only in one respect do they differ, and that is that the majority of cysts are multiple and the level of anæsthesia may not be a safe guide for determining the site of operation.

In the case I report the level of anæsthesia corresponded to the eighth thoracic segment; yet the cyst, or the mass of it, was found at the fourth and fifth segments.

In the Fuerbringer-Hahn case, although the cord was exposed from the second to the seventh thoracic segments, yet at the autopsy the echinococcus vesicles were found to extend the whole length of the spinal canal to the coccyx.

In the Ransom-Anderson case the cysts were headward of the field of operation and were not removed during life.

If pain and sensitiveness of the spine are present, accompanied by tumefaction, they will prove a much safer guide than the level of anæsthesia.

Tumefaction or a distinct swelling upon the back near the spine, accompanied with the ordinary symptoms of spinal cord compression, is a very important symptom of intraspinal cysts, and was noticed in the cases reported by Szekeres (case 3); Lloyd (case 4); Trendelenburg (case 5); Williamson and Tytler (case 8); and in the present instance.

The evacuation of subcutaneous cysts and the demonstration of the scolices or hooklets, occurring coincidently with compression symptoms of the spinal cord accompanied by pain, is an almost sure indication that echinococcus disease of the cord or spine is present, and the case one adapted for surgical rather than medical skill.

Results.

Of the eleven cases reported, seven, or 64 per cent., terminated in recovery. Of the four cases which died, case

No. 1 entered the hospital in December, 1890, and had been under observation for eighteen months preceding his admission. He came under Ransom's care on December 31, 1890. An antiluetic treatment was followed for two weeks. The operation (January 15, 1891) failed to disclose the lesion; the eleventh thoracic to second lumbar segments were exposed. The patient died on the third day following the operation from septicæmia. At the autopsy a cyst the size of a horse-chestnut was found at the tenth thoracic segment; the spinal cord was completely degenerated, and bed sores were present.

The patient in case No. 2 died on the day following the operation. The patient entered the hospital in November, 1890, although three years previously he had experienced violent pains in the back, and had entered a hospital, from which he had been dismissed. He was operated on while in a moribund condition, and nothing but death was to be expected. The autopsy showed complete degeneration of the cord, pleurisy with effusion and hypostatic pneumonia.

In case No. 5 the patient entered the hospital in November, 1895. Cystic tumours began to appear as early as March, 1896. In November, 1896, tapping one of these tumours yielded cystic fluid containing scolices with hooklets. At the spinal operation on November 19, 1896, the whole lower lumbar and sacral cavity were filled with echinococcus cysts. Some improvement followed the operation, but the appearance of new cysts and their removal so weakened the patient that he died nine months after the original operation.

In case No. 7 the patient was a man, aged 27, afflicted with Hodgkin's disease. In the middle of January, 1899, he began to complain of loss of power in the legs. By the end of March, 1899, there was present a complete paraplegia with severe pains, exaggerated reflexes, incontinence, cystitis, and constipation. The patient was first seen by A. F. Wilson and Hale White "at the end of March, 1899," and a diagnosis of pressure on the spinal cord at the level of the second dorsal vertebra was given and laminectomy suggested. The date of operation is not stated; the second,

third and fourth segments of the cord were exposed, and a cyst removed which proved to be a dermoid. The man quickly rallied from the operation, and the wound healed by first intention. The patient was seen eight weeks after the operation. "The pulse and temperature were normal. There was slight occasional voluntary movement in the legs. The exaggeration of the knee-jerks was less, and the rigidity had disappeared. There was almost complete return of all forms of sensation, down to the groins, but no improvement in the lower extremities. The radiating pains had disappeared. The chest moved better. The cystitis and incontinence had disappeared, and the constipation and abdominal distension were less. There was a large sore on the sacrum, and another on the buttock. Considerable as had been the improvement, it appeared to the authors that as much of the tumour had been left behind, and as the pressure symptoms were far from completely relieved, the only chance lay in a further attempt at complete removal. A second operation was performed. A large mass of the tumour was found to have grown backwards through the gap in the spinal canal made at the first operation; the first, fifth, and sixth dorsal laminæ were removed, but still no limit could be seen to the growth. The patient again rallied well, but sank eight hours after the operation."

What might have happened had these cases been operated on sooner is not difficult to conjecture. One, perhaps two, possibly all four of these cases might have recovered. Of the cases terminating in recovery, Szekeres (Case 3) reports that "after the outer sack had been removed and the wound tamponed, the remainder of the sack was thrust out after eight weeks, also a piece of a vertebral arch; this was followed by complete recovery."

Case 4. Dr. Lloyd says: "The after-results have been perfect. The patient has been steadily improving. The dyspnœa, which undoubtedly was due to the hydatids—a symptom that has been reported several times—has entirely disappeared. He is gaining flesh, is less anæmic, has recovered his power over his bladder; and his sexual power, which before the operation had disappeared, is returning. The anæsthesia and analgesia have disappeared, and he is

now able to walk without assistance of any kind, and is ready to return to work. He still complains of some pain, but I believe even this will disappear."

In a personal communication, Dr. Lloyd informed me that the patient died some time afterwards, the result of an accident. The autopsy showed that in all probability the cyst removed from the spinal cord was the original or mother cyst from which all others sprung.

Case No. 6. This patient was briefly alluded to in a lecture by Sir Victor Horsley, and the remark made that "the case is naturally terminating favourably."

The results obtained by Tytler and Williamson (case No. 8) are very gratifying. On December 2, 1902, two years and five months after the operation, the authors note her condition as follows: "The patient is able to walk about the ward, and to walk for a short distance in the street with the aid of one stick. She could perform all the coarse movements of the legs. There was, however, considerable rigidity of the legs, with increase of the reflexes; but sensation was normal. There was no affection of the bladder, and the patient felt quite well."

Dr. Spiller reports on his patient, case No. 9, August 21, 1902, two months after operation, as follows: "Since the operation she has had no pain near the left sacro-sciatic foramen, where the pain was so intense before the operation. The pain in the back has been getting less severe and less frequent. She can stand well on her left limb alone, and her gait is normal. The right lower limb is normal, and entirely free from pain. Resistance to passive movement is about normal in the left lower limb. Patellar reflex is not obtained on the left side, but is normal on the right side. Babinski's reflex is not present on the left side, and the left tendo Achillis reflex is absent. Sensation for pain and touch is normal in the left lower limb. She returned to her home on August 23, 1902. She may be said to be cured; although, of course, we cannot say with certainty that no further pathological condition exists."

Schmidt (case No. 10) comments on his patient as follows: "The relief from pressure symptoms followed rapidly. A few days after the operation an improvement

in the sensory disturbance of the legs was noticed. Four weeks afterwards sensation for touch was everywhere normal, while the sensation for pain was still diminished. Three months after the operation the subjective symptoms were all gone save the girdle sensation, and objectively, the hypæsthetic zone cephalad of the anæsthetic. Now—five months after the operation—these, too, have vanished. Four weeks after the operation the first voluntary movements of the toes were noticed. After five months the patient is able to walk about with a cane. The reflexes are still somewhat exaggerated, and ankle-clonus is still present, also reflex spasms, especially on attempting to walk. The bladder and rectum function normally."

It must be admitted that with the possible exception of my own case, the results obtained have been eminently satisfactory, and these results must surely substantiate that the operation is warranted and even indicated.

Summary.

Intra-spinal Cysts with Operation.

Case.	Operation.	Observer.	Operator.	Situation.	Locality.	Result.
1	Jan. 15, 1891	Ransom	Anderson	10th thoracic	Extra-dural	Death.
2	April 8, 1891	Fuerbringer	Hahn	2nd to 7th thoracic	Extra-dural	Death.
3	Dec. 5, 1893	Szekeres	Rauch	9th to 12th thoracic	Extra-dural	Recovery.
4	June 13, 1896	Meirowitz	Lloyd	8th to 9th thoracic	Extra-dural	Recovery.
5	Nov. 19, 1896	Trendelenburg	Trendelenburg	2nd lumbar to sacrum	Extra-dural	No improvement.
6	Not stated	Gowers	Horsley	3rd lumbar	Extra-dural	Recovery.
7	Not stated	A. D. Tripp	Hale White	1st to 5th thoracic	Extra-dural	Improvement. Recurrence. Death.
8	July 14, 1900	Williamson	Tytler	3rd thoracic	Extra-dural	Recovery.
9	June 30, 1902	Spiller / Musser	Martin	2nd and 3rd lumbar	Intra-dural	Recovery.
10	July 18, 1903	Schmidt	Weber	6th thoracic	Intra-dural	Recovery.
11	Feb. 13, 1905	Krauss	Park	4th to 5th thoracic	Extra-dural	Recovery.

References.

[1] *Brit. Med. Journ.*, November 28, 1891, p. 1144.
[2] *Deut. Zeitsc. f. Chir.*, vol. lxiii., p. 421, Case 1.
[3] *Gyógyászat* (Hungary), August 5, 1894, p. 367.
[4] *Amer. Med. Surg. Bull.*, December 5, 1896.
[5] *Beitr. klin. Chir.*, 1898, vol. xxi., p. 151.
[6] *Clin. Journ.*, London, January 13, 1897, p. 182.
[7] *Brit. Med. Journ.*, March 31, 1900, p. 764.
[8] *Brit. Med. Journ.*, February 7, 1903, p. 301.
[9] *Univ. Penn. Med. Bull.*, March and April, 1903, pp. 27 and 56.
[10] *Deut. Zeitsc. f. Nervenheilk.*, Bd. xxi., Hft. 3.
[11] Read at the thirty-second Annual Meeting of the American Neurological Association, June 5, 1906.

Note by the Editor.—The following cases may be added to the author's list:—

Case.	Operation.	Operator.	Situation.	Locality.	Result.
12	1904	Horsley	5th to 6th thoracic	Extradural	Slight improvement
13	1903	Ballance	2nd to 5th thoracic	Extradural	Recovery
14	Dec. 12, 1895	Horsley	11th thoracic to 2nd lumbar	Extradural	Partial recovery
15	Feb. 22, 1904	Horsley	12th thoracic to 1st lumbar	Extradural	Slight improvement
16	Aug. 7, 1900	Horsley	6th to 8th thoracic	Extradural	Recovery
17	Jan. 27, 1906	Connell	12th thoracic to 1st lumbar	Extradural	Recovery

References.

[12] to [16] Owen, A. G., *Intercolonial Med. Journ. of Australia*, December 20, 1905 vol. x., p. 544.
[17] Connell, E. J., *Intercolonial Med. Journ. of Australia*, March 20, 1906, vol. xi., p. 146.

Review.

An Attempt to Classify Cerebellar Disease, with a Note on Marie's Hereditary Cerebellar Ataxia.

It is unnecessary to insist on the need of some method of classifying primary disease of the cerebellum: it has become almost imperative owing to the confusion which has arisen from the introduction of the term "Hereditary Cerebellar Ataxia" by Marie, as under this title many cases have been described without any regard to the exact nature and the localisation of the pathological lesions.

The numerous published cases of cerebellar disease have been already several times collected together and reviewed, but as the object has been to determine the symptoms due to affection of the cerebellum, or to adduce some facts with regard to its function, all cases have been indiscriminately grouped together without any regard to the nature of the lesions. In the reviews of Nothnagel, Thomas, and Adler [1] little attention was paid to whether the disease was congenital or acquired, or whether it was a primary affection of the functional tissue or a degeneration of this secondary to vascular or interstitial lesions; while Mingazzini [33], who has alone attempted to classify cases according to their morbid anatomy, has, apart from distinguishing congenital from acquired lesions, more or less disregarded the nature of the disease.

As it is essential to base any classification of disease on morbid anatomy and its pathogenesis, only those cases will be included in the present review in which the nature and the extent of the pathological changes were determined by *post-mortem* examination. But even with this restriction it is difficult to classify many of the published cases, owing to the meagre clinical and anatomical records which are available. This is especially so with the cases which were described before more exact methods of examination were in use; many of these must be consequently excluded, as they can be of nothing more than historical interest. The large number of recorded cases of aplasia or congenital lesions of the

cerebellum must be also excluded from this review, but it is not always possible to do so with certainty, as we must rely on the history of the onset and of the course of the disease, as well as on the histological changes found in the central nervous system.

The following mode of classification seems the most natural and the best possible:—

(I.) Primary parenchymatous degeneration of the cerebellum.

(II.) Olivo-ponto-cerebellar atrophy.

(III.) Progressive cerebellar disease due to vascular or interstitial lesions.

(IV.) Acute cerebellar lesions.

In these four groups all cerebellar diseases, apart from tumours and focal lesions of vascular origin, may be included. It will be seen, however, that it is also advisable to discuss the two following groups of cases here; for although in them the cerebellum is not affected, their most prominent symptoms have been described as those of cerebellar disease.

(V.) Degeneration of the spino-cerebellar tracts, the cerebellum being normal or small only.

(VI.) Congenital smallness of the central nervous system associated with cerebellar symptoms.

There are cases which do not fully conform to the type of any of these classes, but when there are main and general resemblances in both the clinical symptoms and the morbid anatomy it seems advisable to attempt to place them in one or other group. This is frequently the most we can do in any branch of medicine, as neither nature nor disease draws lines of sharp distinction.

Class I.—Primary Parenchymatous Degeneration of the Cerebellum.

The cases which I have described in the present number of this Journal [21] seem to be the best examples of primary parenchymatous disease of the cerebellum, with the rest of the central nervous system intact or only secondarily affected.

Fraser's cases [18] probably fall into the same group. A brother and a sister were similarly affected, but there was no other case in the family. The symptoms were present from early childhood and increased slowly in intensity. They were very similar to those in my cases; a reeling and staggering gait,

incoördination of the arms, nystagmoid jerking of the eyes on lateral movement with, in addition, strabismus, which may have been congenital, slow, guttural and hesitating articulation, and tremor of the head in one case at least. Vertigo was prominent in one case, and in both there was primary optic atrophy. The central nervous system of the brother was examined. The cerebellum was reduced to less than half its normal weight, its cortex was little more than half the normal thickness, very few Purkinje cells remained in it, and these were atrophied and shrunken. The white matter of the folia was less affected. The spinal cord, which was examined by the naked eye only, appeared normal, and no other disease was detected in the central nervous system. Fraser's cases have been included among the congenitally small cerebella, but the progressive course of the symptoms, as well as the nature of its morbid anatomy and its familial incidence, make it to my mind most probable that the disease was a primary degeneration of the cerebellar cortex.

In the case of a woman reported by Thomas [64] the symptoms began with slowly progressive affection of gait at the age of 40. There was no similar case in the family; the patient had had syphilis and admitted excessive indulgence in alcohol. She walked on a broad base, reeling from side to side, and there was marked disorder of movement, of the intention-tremor type rather than ataxia, of the lower limbs, but the upper extremities were not affected. All muscles were very hypotonic. Articulation was nasal and scanning, and there was irregular nystagmus on lateral movements of the eyes. Sensation was unaffected, the knee-jerks were brisk and the plantar responses were of the extensor type. She died at the age of 54. The only pathological changes found in the central nervous system were disappearance of the Purkinje cells from many of the folia of the cerebellum, atrophy of these cells in others, though in some they were normal; in the folia from which the Purkinje cells had disappeared there were degenerative changes in the molecular and granular layers, with secondary neuroglial proliferation. There were no vascular or meningeal lesions.

In a case recorded by Mingazzini [33] the cerebellum was very small, its cortex was atrophied and narrow, and the Purkinje cells were diminished in number and in places absent. The white matter and the myelinated fibres of the cortex were also atrophied. The central nuclei were imperfectly developed and the medial portions of the superior peduncles were degenerated.

The middle peduncles and the transverse fibres of the pons were also slightly atrophied. There was, in addition, thickening of the pia mater over the whole central nervous system, and probably as a result of this a slight peripheral degeneration of the cord and atrophy of the cerebral cortex, but these may be regarded as only coincident changes. The patient died at the age of 18. There is no history of the course of the disease, and no similar case in the family. The symptoms and signs were convergent strabismus, nystagmus on movement of the eyes, scanning articulation, rhythmical tremor of the head, slight rigidity of the limbs and ataxia of the intention-tremor type, a reeling and staggering gait with great difficulty in equilibration, and active tendon reflexes. The patient was an idiot and had frequent epileptic convulsions. Mingazzini regarded his case as a partial agenesis and aplasia of the cerebellum, and, though this was most probably its nature, it is cited here as a possible case of cerebellar degeneration.

The cats which were examined by Herringham and Andrews [20] were possibly also instances of primary cerebellar disease. The four kittens of a litter appeared healthy at birth and were for a time able to walk quite naturally. Two of them soon became weak on their legs and gradually incapable of movement, and were destroyed. Later the other two were similarly affected; in these the symptoms were certainly not present from birth. The optic discs and the functions of the cranial nerves were normal; there was marked inability to maintain equilibrium both in rest and in motion: on a smooth surface they attempted to walk on a broad base, reeling and falling from side to side, the defect being chiefly in the movements of the hind limbs. The one kitten was killed at four months, the other at eight months of age, and the central nervous systems were carefully examined. The cerebellum of each was small; the molecular layer of its cortex was only half the normal breadth, the Purkinje cells were scattered all through the granular layer, but they were apparently normal and not appreciably diminished in number; the granular layer was narrow and in places hardly recognisable. The white matter, the central nuclei and the peduncles of the cerebellum, as well as the rest of the central nervous system, were unaffected. The authors regarded the condition as a primary degeneration of the molecular and granular layers of the cerebellar cortex, the distortion of the Purkinje cell layer being secondary to the sclerosis and contraction of the molecular layer. Risien Russell [50] found a somewhat similar condition in the puppy which he examined. All the members of the litter had apparently the

same symptoms, viz., an ataxic and reeling gait and constant oscillation of the head and trunk while at rest, but it is not certain whether the symptoms were present from birth or were acquired. The breadth of the molecular layer was very irregular; in places it was increased, in other places diminished; the granular layer was similar, and the Purkinje cells, which were absent in a considerable portion of the cortex, were in other places heaped up into masses which extended into the granular layer. He regarded the condition as a defective development. Southard [58], too, found a somewhat similar condition in the cerebellum of an epileptic imbecile who had apparently exhibited no symptoms of cerebellar disease during life. The author regarded this condition as a developmental anomaly. The evidence from these cases of Russell and Southard makes it probable that the pathological changes described in the kittens by Herringham and Andrews were also developmental abnormalities, and not degenerative, as these authors assumed. The absence of any secondary degeneration in the white matter of the cerebellum tends to confirm this view. There is evidence that cerebellar symptoms may develop late in life on the basis of congenital abnormalities (see the cases of Nonne and Miura).

Class II.—Olivo-ponto-cerebellar Atrophy.

The second type of primary cerebellar degeneration may be known by this title given to it by Thomas, as the grey matter and fibres of the pons and of the inferior olives undergo simultaneously primary degeneration. It seems to be the form of cerebellar disease which is most easily classified. It is chiefly to Thomas that we owe our knowledge of its clinical and anatomical features. This author published his first case in his well-known thesis on the cerebellum in 1897 [62], he added another case in 1903 [63], and, a third in collaboration with Dejerine [14]. His summary of its main features is, "a type characterised *anatomically* by atrophy of the cerebellar cortex, of the bulbar olives, and of the grey matter of the pons; by total degeneration of the middle cerebellar peduncles, by partial degeneration of the corpora restiformia, and by relative integrity of the central nuclei of the cerebellum; *clinically* by the cerebellar syndrome. It is neither hereditary, familial nor congenital. It comes on at an advanced age and progresses slowly. It falls into the group of primary cell atrophies." This general description covers the three cases, though there were points of slight difference between them. Clinically the most prominent feature was a great defect in equilibration in

standing and walking, to which the typical cerebellar "*démarche ébrieuse*" was due, although the individual movements of the lower limbs were not in the same degree ataxic, and Romberg's sign was absent. In two of the cases the movements of the arms were well coördinated, but in the third there was intention-tremor on movement. Articulation was slow and scanning in all three cases, and nystagmus was observed in two. Anatomically there was slight pallor of the crossed pyramidal tracts of the cord in two of the cases, but the other spinal tracts were normal. The degeneration of the transverse fibres and nuclei of the pons and of the inferior olives was practically complete, and the affection of the cerebellar cortex was very severe, involving all layers, but it was not uniform either in degree or distribution. Emphasis was laid on the very slight amount of neuroglial sclerosis which was found in the degenerated parts. Thomas' second case was complicated by the co-existence of disseminated sclerosis, which, however, the author regarded as independent of the cerebellar disease.

Other recorded cases have considerable resemblance, both clinically and pathologically, to this type, and may be included in it. That published by Pierret [45] was probably identical; but if the history was trustworthy the symptoms appeared at a much earlier age—4 years—and there was much more tremor and incoördination of the upper extremities. Pathologically there was no essential difference, except a considerable amount of secondary sclerosis.

The clinical course of Royet and Collet's case [49] was typical except that there were contractures, but the cerebellum, which was small and appeared tough and sclerosed, was not examined microscopically. The degeneration of the pons and olives was very marked, and the spinal cord was normal.

The case reported by Arndt [3] differed from the type chiefly by the relatively slight affection of the cortex, in which there was only diminution and atrophy of the Purkinje cells, and by the more intense degeneration and sclerosis of the white matter of the cerebellum; the pons and the olives were completely degenerated. There was also slight affection of the pyramidal tracts. The author believed the degeneration of the nervous elements and the glial sclerosis were secondary to vascular disease; is seems, however, more probable that the definitely systematised degenerations which he described were independent of the vascular disease that was present, especially as the central nuclei and the superior cerebellar peduncles escaped. The symptoms began with vertigo and uncertainty of gait at the age of 66, four years before death.

The patient's gait was reeling and drunken, and his base broadened by separation of his feet. There was also slight dysarthria, paresis of the limbs without rigidity, and irregularity of the volitional movements, which were more or less of the intention-tremor type.

There was no accurate clinical observation of the case described by Redlich [47], but the pathological changes were very similar to those in Arndt's case. He regarded the case as a primary disease of the cerebellum, the Purkinje cells being the portion of the cortex which was first affected.

The subject of the recent publication by Schweiger [53] was a demented woman who died at the age of 58. Weakness of the legs and tremor of the upper extremities developed at the age of 51, and two years later articulation became affected. When she came under observation at 57 there was nystagmus, scanning speech, intention-tremor and slight spastic paresis of the arms, more marked spastic paralysis of the legs, paralysis of the sphincters, increased knee-jerks and ankle-clonus. The clinical diagnosis was disseminated sclerosis. The lateral lobes of the cerebellum were severely affected, the vermis less so, while the tonsils were normal. The central white matter of the lateral lobes had suffered the most severely, and that of the folia was also intensely degenerated. There was considerable atrophy of the granular layer of the cortex, the Purkinje cells were reduced in number, and those which remained were atrophied, but the molecular layer was almost normal. The inferior olives and the olivo-cerebellar fibres, as well as the middle cerebellar peduncles, the transverse fibres of the pons and the pontine nuclei, were completely degenerated. The dentate nuclei and the superior cerebellar peduncles were intact, but there was probably some affection of the roof nuclei. The case is remarkable owing to the degeneration of the cortico-pontine tracts, but the cortico-spinal tracts were normal. In the middle thoracic cord there was a focus which involved only the white matter and destroyed all its fibres, but the only secondary degeneration which resulted was of the distal part of the pyramidal tracts. This lesion was regarded as a patch of disseminated sclerosis, but it was not sclerosed; if this was its nature it must have been quite recent. I find it difficult to accept the author's interpretation of the pathological changes in this case. According to his view the original lesion was as sclerotic process, similar to the spinal lesion, of the central white matter of the lateral lobes of the cerebellum; the affection of the cerebellar cortex and of the pontine and olivary

systems was secondary to this. The degeneration of the pons and olives was so complete as to necessitate the conclusion that the primary lesion must have occurred in very early, or even in fœtal, life. He admits that there was no histological evidence of the primary encephalitic process which he postulates, but adds "nur so ist das Erhaltenbleiben der Purkinje'schen Zellen zu erklären." But on the contrary, if axones had been destroyed by an encephalitic process, the chief change would have been found in the cells from which those axones spring, and the Purkinje cells are the chief elements of the cortex which send axones into the central white matter. Further, it would be very remarkable if encephalitic foci had involved the two sides of the cerebellum symmetrically, and had, although seated in the central white matter, spared the dentate nuclei. From these facts it seems more probable that the olivo-ponto-cerebellar disease was of the type described by Thomas, and that the spinal lesion was a coincidence only.

Two cases in which, in addition to olivo-ponto cerebellar atrophy, there was tract degeneration in the spinal cord may be most naturally referred to here. The one was published by Menzel [31], the other by Thomas [62], (Obs. V.). There was probably a familial incidence of the disease in Menzel's case; the mother, who died at 60, had tremor of the head and uncertain gait; five of her seven children had evidences of nervous disease, and of these two sisters and a brother were probably affected similarly to Menzel's case. One of his daughters, too, had some form of hereditary ataxia. In the patient himself the symptoms appeared at 28 and progressed slowly. His gait was very unsteady and reeling, as well as slightly spastic; the volitional movements of his hands were wildly ataxic, and his upper extremities were feeble and rigid; his speech was slow and badly articulated, and there were involuntary movements of his head (probably a tic) and facial muscles. The knee-jerks were exaggerated. He died at the age of 46. In addition to degeneration of the cerebellum, pons and olives, the dorsal columns of the cord and the cortico-spinal and spino-cerebellar tracts were degenerated. The pathological changes in the cord were thus those of Friedreich's disease.

In Thomas's case [62] there was a neuropathic heredity, but no other case of the disease in the family. The symptoms were complicated by the presence of hysteria. They commenced at the age of 25 with progressive feebleness of the limbs, failure of vision, a staggering gait and intention-tremor of the arms;

articulation became slow and scanning. In addition to atrophy of the cerebellum, pons and olives, which was similar to but not so severe as in the other cases of olivo-ponto-cerebellar atrophy, there was degeneration of the dorsal columns and of the spino-cerebellar tracts of the cord, and some atrophy of the pyramidal tracts, especially in the lumbo-sacral segments. Thomas regarded the cerebellar lesion in this case as a secondary atrophy of peripheral origin, the initial lesion being atrophy of the nuclei of the principal afferent tracts of the cerebellum, especially of the grey matter of the pons.

Class III.—Progressive Cerebellar Disease due to Vascular or Interstitial Lesions.

Although diffuse vascular or interstitial lesions of the cerebellum are often diagnosed, there is very little pathological evidence that they occur frequently. In Schultze's case [52], which was attributed to alcoholism, the onset occurred at 39. Gait became drunken and cerebellar in type, but Romberg's sign was absent; articulation was indistinct and slow, and there was intention-tremor of the arms and slight nystagmus. The symptoms progressed rapidly, and death took place at the age of 43. There was considerable atrophy and sclerosis of the cerebellum and brain-stem. The Purkinje cells of the cerebellar cortex were very scanty, and those which remained were atrophied; the molecular and granular layers were less affected. The lesions tended to be focal and very diffuse, and there was a large amount of secondary neuroglial sclerosis. The central nuclei were very atrophic, and the superior peduncles degenerated. The degeneration of the olives and of the transverse fibres of the pons was probably secondary to the cerebellar disease. The spinal cord was normal, excepting for slight pallor of the pyramidal tracts.

Michell Clarke [10] has published, as a case of sclerotic atrophy of the cerebrum and cerebellum of familial type, the history of a boy who was healthy till 7, when the symptoms began with choreiform jerkings of the limbs. Later, vision failed, the limbs became ataxic and spastic, the knee-jerks were exaggerated, and the plantar reflexes of the extensor type. Articulation was indistinct, and there was progressive mental deterioration. Two maternal uncles had been affected with the same symptoms, the onset occurring at the same age. *Post-mortem* examination revealed atrophy of the cerebral cortex, sclerosis of the white matter of the forebrain, and complete degeneration of

the cortico-spinal tracts. The cerebellar cortex was but little affected, but there were numerous patches of sclerosis throughout the white matter of the cerebellum.

Southard [58], in a congenitally syphilitic negro who died at the age of 23 with a history of progressive incoördination of the movements of the limbs, tremors, nystagmus and mental deterioration, found sclerosis of the cerebellum, absence of the Purkinje cells in many of its folia, and atrophy of its molecular and granular layers, in addition to chronic leptomeningitis and focal sclerosis of the forebrain. The medullæ of the folia were much degenerated, and there was an extreme amount of gliosis in the cortex, white matter, and central nuclei.

Finally Catolo [9], in a case which is difficult to interpret, found marked atrophy of the brain-stem and cerebellum, with diminution of the Purkinje cells of the cerebellar cortex and secondary sclerosis in their place, as well as patches of sclerosis without secondary degeneration throughout the cerebellum, some of them involving the dentate nuclei. Similar patches were found in the brain-stem. There was also partial atrophy of the olives and pons, and pseudo-system degeneration of the ventro-lateral columns of the cord. The vessels of the central nervous system were much diseased. The clinical symptoms, which were progressive, appeared at the age of 38 after an attack of cholera. They were nystagmus and diplopia, tremor of the head, intention tremor of the limbs, an ataxo-cerebellar and spastic gait, scanning speech and exaggeration of the tendon reflexes. The author regarded the case as one of disseminated sclerosis associated with cerebellar atrophy, the latter being probably due to the vascular disease.

Class IV.—Acute Cerebellar Lesions.

Batten [5] and others have attributed the cases of acute ataxia which occur relatively frequently in children during and after infective illnesses to lesions of the cerebellum. The pathological changes in several of these cases have been studied, but, with the exception of a case reported by Nauwerck [38] as cerebellar encephalitis following influenza, histological examination was possible only at long periods after the onset. It seems probable that while in some of the cases the disease is an inflammatory lesion of the nature of an encaphalitis, in others it is an acute degeneration of the nervous elements of the cerebellar cortex due to the action of toxins.

Clapton's case [11] is an excellent example. A healthy child

of 4 suddenly became unable to use her limbs or even to talk, after an attack of measles. She apparently remained more or less in this state for about six months; from that age improvement gradually set in, but till about 15 she could not use her hands properly and walked unsteadily. She died at 33. The cerebellum was less than a third of its normal weight, and on microscopical examination it was found that there was almost complete absence of the proper nerve substance of its cortex and great increase of neuroglia.

Hammarberg's case [19] was very similar. The onset was acute, with an attack of Hirnentzündung—"brain fever"—at the age of 7. For the first three months there was constant oscillatory movement of the head and limbs, and speech was lost. As these symptoms disappeared all volitional movements became wildly ataxic, articulation was scanning, gait reeling and uncertain, and there were choreiform movements of the fingers. Death occurred at 24. The pathological changes in the cerebellum were very similar to those in Clapton's case, but sclerotic foci were also present in the medulla and cerebrum.

The cases recorded by Huppert [22] and Sommer [57] were also similar. In each, at the age of 3 years, there was some form of acute brain disease, and thereafter gait was drunken and reeling, and there were other symptoms of cerebellar disease. Both children become idiots. The cerebellum of each was small and evidently sclerosed. In Sommer's case, of which there is a good description, the affection was not uniform; in the most affected parts all layers of the cortex were extremely atrophied, the Purkinje cells had disappeared, and the white matter was reduced and sclerosed.

Bond's case [7] was probably similar, but the history of the disease was not definite enough to decide with certainty if the onset was acute, or even if it was post-natal.

In the case which was examined pathologically by Spiller [59] the onset occurred apparently during an attack of scarlet fever at the age of 3 years, but the fact that the child was late in learning to walk raises the suspicion that the lesion may have been congenital. There was a gradual but not complete recovery from the symptoms; gait always remained staggering. Death occurred at 19. The cerebellum was small and widely affected by sclerotic foci, some of which extended sufficiently deep to reach the dentate nuclei. No cortex was present in the sclerotic areas; elsewhere its different layers were reduced in thickness. The cerebellar peduncles were small, and the olives and pons very atrophic.

The spinal cord was normal, but one cerebral hemisphere was under-developed and the corpus callosum was deficient.

In a case described by Sepilli [56] there was an acute onset during convalescence from an attack of typhoid fever, at the age of 28. The symptoms when the patient was later observed were general tremor and choreiform movements of the arms, ataxia of all voluntary movements, a reeling and drunken gait, and some affection of articulation. After death, which occurred four years after the onset, it was found that the cerebellum was very small, its cortex was extremely atrophied, and practically all its Purkinje cells had disappeared; the white matter of the folia contained very few myelinated fibres, and the dentate nuclei were also affected.

In the cat examined by Krohn [25] the history of acute onset was very definite. It occurred at the age of three months, when the cat suddenly became paralysed. Later, on attempting to walk, the animal reeled from side to side: "the hind limbs seemed to wish to go faster than the fore, giving a ludicrous effect." The movements of the limbs were ataxic, and there was considerable tremor, especially of the head. When the cat was killed at 11 months of age the cerebellum was found small and firm, its cortex was reduced to scarcely more than half its normal thickness, the molecular layer being the most severely affected, and the Purkinje cells were almost quite absent. The white matter was almost normal.

In the majority of these cases with acute onset the disease seems to have consisted of diffuse or focal lesions of the nature of encephalitis—that is, primary disease of the interstitial substance or of the vessels; but in the cat observed by Krohn, as well as in the two following cases, it was more probably a primary cell degeneration due to the action of toxins.

In the case recently recorded by Rossi [48] as primary parenchymatous atrophy of the cerebellar cortex, the symptoms appeared at the age of 59 after an attack of acute diarrhœa with pains in, and marked feebleness of, the limbs, and great disturbance of articulation. After a time there was some recovery, but later slight progression of the symptoms. Gait was staggering and of the cerebellar type, yet also somewhat spastic; there was asynergia and intention-tremor of the legs without any loss of power, articulation was slow and lisping, but there was no nystagmus. There was some analgesia of the legs and the plantar responses were extensor in type, neither of which can be regarded as cerebellar symptoms. Death occurred at 68. The cerebellum

was considerably reduced in weight and in volume, but the disease was practically limited to its upper surface. In the affected folia the molecular layer was much reduced in thickness; the Purkinje cells were completely absent, and the granular layer had lost many of its cells, but the white matter was little affected. In the other parts there was only a numerical reduction of the Purkinje cells. The central nuclei and cerebellar peduncles, as well as the rest of the central nervous system, were normal.

In connection with the etiology of the cortical degeneration in Rossi's case, it is interesting that Murri [37] has reported a case in which the Purkinje cells degenerated after an attack of enteritis, owing presumably to the action of toxins of intestinal origin. The clinical symptoms were those of cerebellar disease.

Lüthje [27] has insisted that the majority of these cases of acute ataxia which come on during or after the infective diseases, are due either to pure cerebral or diffuse cerebrospinal lesions. He cites the case of Ebstein's patient [15] who, when 48, during an attack of enteric fever, developed ataxia and paresis of the limbs, tremor of the head and limbs, and faulty articulation. The symptoms gradually diminished. He died eight years after the onset. The autopsy revealed diffuse focal perivascular and meningeal lesions throughout the cord and medulla; the cerebrum and cerebellum were normal.

Class V.—Degeneration of the Spino-Cerebellar Tracts, the Cerebellum being normal or small only.

The justification for including these cases among, or rather contrasting them with, those in which the cerebellum itself is affected, is in the clinical experience that their prominent symptoms are similar to, or may be identical with, those of cerebellar disease. Attention has been directed to this by the experimental work of Marburg [28] and Bing [6], who observed marked cerebellar symptoms after section of the spino-cerebellar tracts in the cord. A further reason for considering these cases here is the fact that some of the cases originally included by Marie in *Hérédo-ataxie cérébelleuse* belonged to this group.

(1) Cases which do not belong to any well-recognised type of disease may be considered first.

Klippel and Durante's observations [24], which were subsequently completed by Londe [26], Vincelet [66], and by the authors who have described the anatomical examination of the central nervous systems, were made on three members of a

family, of which the mother and one of her sisters were apparently similarly affected. The onset occurred between the ages of 25 and 35 years, in two of the cases with cramps, violent pains and numbness in the legs, followed by a slowly progressive affection of gait, which was described as "démarche titubante," and Romberg's sign. The face was immobile, articulation hesitating, indistinct, and scanning; coarse nystagmoid jerking was observed on volitional movement of the eyes, and in two cases there was slight failure of vision, apparently without any disturbance of the pupillary reactions (on this point there are discrepancies between the reports of the different observers who published clinical details of the cases). Fibrillation in the limbs and facial muscles, and frequent intermittent muscle cramps which occasionally interfered very much with volitional movement, were prominent symptoms; it was to these cramps that Klippel and Durante attributed the disorders of movement of the different parts of the body. In addition there was rigidity and slowness and feebleness of movement and easy fatigue, but no true ataxia of either the upper or lower limbs. Contractures finally developed and produced deformities. In all three cases there was considerable diminution of cutaneous sensation, especially to heat and cold, on the legs and feet. The knee-jerks were normal or slightly exaggerated in two of the cases, though in the eldest brother they were diminished and finally lost. In the only case in which the state of the plantar reflexes was recorded, they were extensor in type. The three patients have died, and their central nervous systems have been examined by Thomas and Roux [65], Switalski [61], and Rydel [51]. The pathological changes found in all were so similar that they may be described together. The central nervous system was small, especially the cord, brain-stem, and cerebellum. The dorsal columns and the dorsal and ventral spino-cerebellar tracts of the cord were degenerated, and Clarke's column had lost most of its cells and fibres. There was also slight atrophy of the grey matter of the ventral horns. The only abnormality in the brain-stem was degeneration of the spino-cerebellar tracts, except in Switalski's case, in which the middle cerebellar peduncles and the ventral part of the pons were small. No histological change was found in the cerebellum. Thomas and Roux, in discussing the significance of the small size of the central nervous system, point out that though the age at the onset of the symptoms was in favour of it being due to a regressive atrophy, the absence of secondary degeneration and sclerosis favours the view that it merely represented incomplete development.

The chief clinical features of the twenty-one cases which Sanger Brown [8] observed in four generations of the one family were: onset, most frequently between the ages of 16 and 35, with ataxia of the legs and later of the arms, facial, ocular, head, laryngeal and pharyngeal muscles. The gait was described as a cerebellar reeling; there was no tendency to Romberg's sign. Later in the illness, palsies and permanent spastic contractures developed. There were also involuntary choreiform movements of the head and limbs "whenever it was attempted to maintain these parts in a fixed position by volitional effort." Optic atrophy was observed, with deficient pupillary reaction to light. Sensation was unaffected, the tendon-jerks were exaggerated, and there was no tendency to deformities, save those produced by the spastic contractures. Three of these patients have since then been examined pathologically, one by Meyer [32], and two by Barker [4], and in each of the three the same changes were found in the nervous system. The cerebrum was well developed and normal; the cerebellum was only very slightly, if at all, smaller than natural, and was normal in structure, except, perhaps, for slight diminution in the number of the Purkinje cells; in the brain-stem there were only insignificant changes. In the spinal cord, on the other hand, there were pronounced alterations; it was small in proportion to the rest of the central nervous system; the dorsal spino-cerebellar tracts and Clarke's column were completely degenerated; Gowers' tract was slightly affected, while in the dorsal columns there was degeneration of the middle root zones with ascending degeneration of Goll's columns.

In Perrero's case [44] there was no heredity, but from the age of 2 years the patient staggered and had tremor of the head. From the sixth year there was kypho-scoliosis, pes equino-varus, and a gradual deterioration of the power of walking. The anatomical change was enormous atrophy of the cerebellum and cerebrum, which was probably congenital, as microscopical examination revealed only the character of simple atrophy. In the spinal cord there was degeneration of the dorsal and ventral spino-cerebellum tracts, diminution of the cells of Clarke's column and degeneration in the dorsal columns. The special interest of this case is that, like Menzel's and others, it is regarded as forming a link between Friedreich's disease and hereditary cerebellar ataxia.

(2) Friedreich's disease is the best recognised type in which there is system degeneration of the spino-cerebellar tracts; a

short reference is necessary to the part the cerebellum plays in the production of its symptoms, owing to the repeated attempts to establish its identity with hereditary cerebellar ataxia—(Raymond [46]) or to insist that the two diseases are linked by intermediate forms—(Nonne [41], Londe [26], Seiffer [54]). To me it seems that the question needs little discussion, as Marie's disease, by which a primary atrophy of the cerebellum is generally understood, cannot be regarded as a disease in the ordinary acceptation of the term in modern medicine; the cases which have been included in it are not distinguished by constancy of their clinical symptoms, morbid anatomy, or pathogenesis.

Senator [55] some years ago argued, in opposition to Schultze, that the symptoms of Friedreich's disease were due to concomitant spinal and cerebellar lesions, but without any definite proof of his assertion. More recently, Phillipe and Oberthur (cited by Raymond) found in a case of Friedreich's ataxia general atrophy, without sclerosis of the cerebellar cortex and white matter, of the inferior and middle cerebellar peduncles, and of the transverse fibres and nuclei of the pons. The only other reference to lesions in the cerebellum in Friedreich's ataxia with which I am acquainted is by Mott [35], who found, in a patient who died twenty-one years after the onset of the symptoms, atrophy of the nuclei dentati and of the Purkinje cells of the cortex, associated with severe vascular degeneration, which was probably the causal factor. In two advanced cases in which I have examined the cerebellum by modern methods I failed to find any definite evidence of disease; here and there the Purkinje cells were placed rather far apart, but it is known that their arrangement in the normal brain is often irregular. It seems, then, probable that the pathological changes in the cerebellum and its peduncles which Phillipe and Oberthur found in the case of Friedreich's disease which they examined, can be regarded only as an exceptional coincidence, and there is thus very little justification for Raymond's assertion of the identity of this definite disease with the ill-defined groups of cases which have been described under the title "Hereditary Cerebellar Ataxia." Even if lesions of the cerebellum are found in Friedreich's disease they may be only secondary to the degeneration of the spino-cerebellar tracts which terminate in its cortex. In cases of combined system degeneration of the cord, in which the spino-cerebellar tracts degenerate, I have found foci of degeneration of all layers of the cerebellar cortex. It seems possible that this change was secondary to degeneration of the cerebellopetal fibres; on the other hand

it may have been merely the result of the direct action of the toxins to which the spinal degenerations are generally attributed. This view is made probable by the fact that Sträussler [60], whose recent paper is confirmatory of the observations of Weigert and Raecke, found similar degenerative changes in the cerebellum in general paralysis, which he attributed to the action of toxins circulating in the cerebrospinal fluid. Although there is no positive evidence that atrophy or degeneration of the cerebellum may follow degeneration of the spino-cerebellar tracts, it seems probable from analogy, as the cerebellum is very prone to undergo regressive changes as a result of cerebral lesions. The atrophy may travel by the superior peduncles, as probably occurred in the case of Mott and Tredgold [36], in which the molecular and granular layers of the cortex were atrophied and all Purkinje cells had disappeared from some of the folia; or through the pons and middle peduncles, as in the cases of Ferrier [16] and Cornelius [12], in which similar changes were observed in the cerebellar cortex. In the majority of these cases the primary cerebral lesion was congenital or occurred early in life, and the cerebellar changes were consequently regarded as due to arrest of development; but in the case described by Ferrier [16] the cerebral lesion occurred at the age of 30, and Cornu [13] has observed crossed cerebellar atrophy due to destruction of the contralateral basal ganglia by a tumour which had given rise to symptoms for only fifteen months before death. These cases indicate that this form of cerebellar atrophy is not merely a condition of arrested development, but is due to active regressive changes.

Class VI.—Cerebellar Symptoms associated with Congenital Smallness of the Central Nervous System.

This class is represented by the familial cases described by Nonne [40] and Miura [34]; the former were originally included by Marie in his paper on "Hereditary Cerebellar Ataxia."

Nonne recorded the cases of three brothers in whom the onset of the disease occurred at the ages of 10, 14, and 30. The first symptom to appear was awkwardness and uncertainty of gait, which increased slowly; Romberg's sign was absent. Articulation became loud, nasal and explosive, and was accompanied by excess of facial movement. There was optic atrophy with contraction of the visual fields, and various ocular movements were limited in range and interrupted by nystagmoid jerkings of the eyes. The movements of the limbs were ataxic, and there was

difficulty in relaxing contracted muscles, but no paresis. Sensation and the reflexes were unaffected. There was marked mental deterioration during the course of the disease. The pathological changes in one of the cases was described in Nonne's original paper, and since then the central nervous system of another has been examined [41]. In both the condition was the same. The whole central nervous system was small, the weight of the cerebellum and brain-stem was less than three-quarters of the normal. In one case the spinal cord was normal in size, in the other only equal to that of a boy of 10 years. No definite disease could be found in any part of the nervous system; the symptoms could consequently only be regarded " als Ausdruck einer mangelhaften Anlage, speciell des Kleinhirns."

Miura has described the cases of two brothers; their mother and two maternal cousins were probably similarly affected. The disease began with unsteadiness in walking, in the one at the age of 25, in the other at 33. Later, gait became reeling and ataxic, but Romberg's sign was absent. Vision became dim, and articulation explosive and stuttering. The facial expressions were remarkably fixed and stupid. Nystagmus was observed in one case, in the other it was absent. The limbs were tremulous, very ataxic in movement, and their strength was poor. Sensation and the reflexes were intact. The nervous system of one of the brothers was examined. The cerebellum and brain-stem were small; the former, which was little more than half its normal weight, appeared sclerosed, but no histological abnormality could be detected on microscopical examination. The cord was also small, but its tracts were normal. The clinical and anatomical similarity to Nonne's cases is obvious; and, as in those cases, the symptoms can be ascribed to a developmental defect only. But it is difficult to see why such prominent symptoms should first appear in early manhood and progress to the total disablement of the patient, if they were due only to a congenital deficiency to which neither degenerative nor regressive changes were superadded. More especially is this so with regard to the symptoms which were referred to the cerebellar deficiency, when it is remembered that with many cases of congenitally small or deformed cerebella, in which the disease was either bilateral (as in the cases of Otto [43], Ingels [23], Major [30], and probably in Ferrier's [17]) or unilateral (Neubürger and Edinger [39], Nonne [42]), no abnormal symptoms were associated; and that in other cases in which symptoms of cerebellar disease were present these gradually diminished with the increase of age (Anton [2]).

"Hereditary Cerebellar Ataxia" of Marie.

It will be remembered that Marie [29] attempted to define a new clinical type under this title by collating the cases which had been published by Fraser, Sanger Brown, Nonne, and Klippel and Durante, and that when his paper was originally published he himself had not observed any case which he could include in it. At that time the nature of the disease had been determined by *post-mortem* examination in two only of these cases.

The justification for Marie's definition of this new type must be therefore sought, in the first place, in the subsequent history and further analysis of these cases, and, secondly, in our clinical experience of its utility.

It must be again insisted that the classification of disease must be based on morbid anatomy and pathogenesis, and not on clinical symptoms alone. The constant association of a set of symptoms or physical signs may justify classing cases together provisionally under a single term, but only so long as it is impossible to determine accurately the pathological changes to which they are due. Consequently, in a discussion on "Hereditary Cerebellar Ataxia," only those cases in which a *post-mortem* examination has been made must be considered.

But an analysis of the clinical records of the cases which Marie orginally collected together shows that the symptoms of even these cases were by no means identical or even similar. The disease was hereditary in the cases described by Sanger Brown and by Klippel and Durante, but there was no similar case in the ascendant or collateral lines of the families observed by Fraser and by Nonne. Thus the term "hereditary" was not entirely justified; the disease was undoubtedly familial, and there was consequently in all probability an hereditary tendency to it, but this does not justify the use of the adjective.

One of the points of difference which has been adopted in many text-books between Marie's disease and Friedreich's ataxia, is that in the former the symptoms first appear in early adult life; but in Fraser's cases they were present from early childhood, and in some of Sanger Brown's they were observed as early as the eleventh year.

Finally, many of the symptoms exhibited by one or other of these patients could not be referred to disease of the cerebellum; for instance, the severe lancinating pains, the anæsthesia, and the inactivity of the pupils to light which were present in Klippel and Durante's cases. Nor could disease of the cerebellum explain the

intermittent spastic contractions of the muscles which were a prominent symptom in these cases, and to which these authors attributed the other abnormal symptoms. Optic atrophy, another symptom of " Hereditary Cerebellar Ataxia," could be regarded as only a coincident dystrophy; it was observed in some instances only.

But any justification there may have been for including the cases originally collated by Marie under the title " Hereditary Cerebellar Ataxia," has been shattered by the anatomical examination of the central nervous system. The morbid anatomy of the disease which affected the different families has been now determined in one or more members of each family, and has been already described in this review. In the cases of Klippel and Durante, and of Sanger Brown, the structure of the cerebellum was normal, and this organ was at the most only very slightly smaller than natural. In the spinal cord, on the other hand, there was degeneration of the dorsal columns and of the spino-cerebellar tracts. In Nonne's two cases no pathological changes were found, but in both the cerebellum was unnaturally small, and in one of these the whole central nervous system was considerably below the normal size. Only in Fraser's case was there the slightest evidence of acquired cerebellar disease.

Nor does a review of the large amount of literature which has appeared on " Hereditary Cerebellar Ataxia " since Marie's original publication help to justify its existence. The majority of the cases of progressive cerebellar disease which have been published belong to the class of the olivo-ponto-cerebellar atrophies, and in not a single one of these were the symptoms either hereditary or familial, and in almost all of them the onset occurred in advanced life. The same points of difference separate Thomas' and Rossi's cases from Marie's hypothetical group. As far as I am aware, no other case of cerebellar disease, with the exception of those published by myself [21], has been recorded in which the symptoms resembled those of Marie's syndrome, and in which the disease has been limited to the cerebellum.

We are consequently driven to the conclusion that no form of disease exists to which the term " Hereditary Cerebellar Ataxia " can be aptly applied. This title has been a convenient pigeon-hole in which to group together cases of obscure nature with some symptoms in common, and it may have been of service in drawing attention to such cases till it was possible to classify them accurately; but neither clinical nor pathological experience justifies its retention as the descriptive title of a form of disease.

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GORDON HOLMES.

PUBLICATIONS RECENTLY RECEIVED.

[*Notes on a book under this heading do not preclude a subsequent heading.*]

The Borderland of Epilepsy. By Sir WILLIAM R. GOWERS. Pp. 121. London: Churchill, 1907.

This is an interesting reprint, with additions, of the lectures published in the *Lancet* and *British Medical Journal*. It deals with various paroxsymal manifestations not definitely epileptic in origin. It is written with all Sir William Gowers's accustomed ease, and the chapter on "Vagal and Vaso-Vagal" attacks breaks new ground.

La Couche Optique—Le Syndrome Thalamique. Par le Dr. GUSTAVE ROUSSY. Pp. 371. Paris: Steinheil, 1907.

The author has gathered together a series of experimental and clinical observations on lesions of the optic thalamus. He gives unusually full descriptions of the pathological findings in the two cases reported by Dejerine and Egger, and to these he adds two others which came to autopsy. The "syndrome thalamique" consists of: (1) a persistent partial hemianæsthesia; (2) slight hemiplegia, with (3) ataxy and astereognosis of the same half of the body; (4) pains, and (5) choreo-athetotic movements on the hemiplegic side. The book is admirably arranged and lucidly written.

On the Functions of the Cerebrum: The Frontal Lobes. By SHEPHERD IVORY FRANZ, *Archives of Psychology*, No. 2, March, 1907. New York: The Science Press.

An experimental study of the functions of the frontal lobes in cats and monkeys. These animals were taught simple tricks, such as how to open the door of a cage. Then the frontal lobes were destroyed and the recently formed habits were lost. Such loss was not caused by lesions of other portions of the brain. Habits once lost may be relearned, and long-standing habits seem to be retained. A plea is entered for a more careful mental analysis of cases of brain disease in man.

Die Geschwülste des Nervensystems. Von Prof. Dr. LUDWIG BRUNS. Zweite gänzlich umgearbeitete Auflage mit 64 Abbildungen im Text. Pp. 480. Berlin: S. Karger, 1908.

A second edition of this well-known work, which first appeared in 1897. It is essentially practical, and the diagnostic significance of symptoms and signs is particularly emphasised.

Vorlesungen zur Einführung in die experimentelle Pädagogik und ihre psychologischen Grundlagen. Von ERNST MEUMANN. Bd. 1. Pp. 555. Leipzig: Wilhelm Engelmann, 1907.

This volume contains the first ten of a series of lectures on the psychological basis of pedagogy. Most of these lectures deal with the development of the psychical aptitudes, such as memory, speech, &c. In the second volume the author intends to deal with special problems, such as reading, drawing, and reckoning.

Writers of "Original Articles and Clinical Cases" are supplied free of charge with 50 copies reprinted in the form in which the paper stands in the pages of "Brain." If reprints are required in pamphlet form, with wrapper, title-page, &c., and re-numbered pages, they must be ordered, at the expense of the writers, from Messrs. BALE, SONS & DANIELSSON, Ltd., 83-91, Great Titchfield Street, London, W.

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EDITOR.

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A JOURNAL OF NEUROLOGY,
FOR
VOL. XXX., 1907.

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AND

LIST OF OFFICE BEARERS AND MEMBERS

OF THE

Neurological Society of the United Kingdom

1907

WITH THE

Annual Report of Council, and Balance Sheet for the Year 1906

London
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1907

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Neurological Society of the United Kingdom.

REPORT OF THE COUNCIL FOR 1906.

FEBRUARY 21st, 1907.

The Council has much pleasure in reporting the continued prosperity of the Society, which now numbers 256 members.

The Society regrets the loss by death of four of its members: Dr. Andriezen, Dr. Syers, Dr. Bryant and Dr. Schorstein.

The Presidential Address was given on November 1, 1906, by Sir Thomas Barlow, Bart., "On Some of the Nervous Complications of the Specific Fevers," and was fully reported in "Brain." At the first meeting of the year, held on February 1, Mr. W. Bateson, F.R.S., read a paper "On Mendelian Heredity and its Application to Man." A summer meeting was held on Saturday, July 14, at the National Hospital, and was well attended both by metropolitan and extra-metropolitan members.

The Hughlings Jackson Lecture was delivered by Sir Victor Horsley, F.R.S., on November 29, "On the Illustration by Recent Research of Dr. Hughlings Jackson's Views on the Functions of the Cerebellum."

One subject meeting on ocular palsies, and three ordinary meetings of the Society were held; the abstract of the Proceedings shows the scope and character of the work of the Society during the past year.

The question of amalgamation of the Society with the Union of Medical Societies came again before the Society in an amended form, and at a Special Meeting held on December 6, 1906, the following resolution was passed: "That the Neurological Society of the United Kingdom agrees to take part in the Amalgamation of Medical Societies and appoints Dr. Head as its delegate to the new Committee."

From a perusal of the balance sheet it will be seen that the finances of the Society are in a satisfactory condition.

Proceedings of the Society during the Year 1906.

February 1.—Paper by Mr. W. BATESON, F.R.S., on "Mendelian heredity and its application to man."

March 1.—Pathological Meeting. Paper by Dr. L. S. DUDGEON, on (1) "Acute diphtheritic toxæmia, with special reference to acute cardiac failure." (2) Lantern Demonstration by Dr. GORDON HOLMES and Mr. J. H. PARSONS, on "The pathological anatomy of amaurotic family idiocy."

March 29.—Clinical Meeting. Cases illustrating varieties of ocular palsies. Mr. MAYOU, "Congenital double ptosis with defective upward movement in both eyes, and concomitant convergent strabismus." Dr. RAYNER BATTEN, "Congenital paralysis of left external rectus." Dr. C. MARCUS, "Two cases of partial iridoplegia, with peculiar pupil phenomena in a boy aged 12 and in a woman aged 32." Dr. POYNTON, "Right ocular paralysis and right hemiplegia." Dr. BERTRAM ABRAHAMS (*a*), "Unilateral paralysis of third nerve." (*b*) "Bilateral partial paralysis of third nerve." Dr. OTTO GRÜNBAUM, "Recurrent third nerve paralysis." Dr. RAYNER BATTEN, "Loss of upward movements, limitation of downward movements, partial iridoplegia, optic neuritis." Dr. GEORGE JOHNSTON, "Myasthenia gravis with double ptosis and diplopia." Dr. S. A. K. WILSON, (*a*) "Case of Friedreich's ataxia with, among other eye symptoms, weakness of downward conjugate movements." (*b*) "Case of migraine ophthalmoplégique." (*c*) "Case of disseminated sclerosis with weakness of each internal rectus, and nystagmus on lateral deviation, limited to the outer eye." Mr. J. H. PARSONS, (*a*) "Bilateral nuclear sixth paralysis in a case of (?) Friedreich's disease." (*b*) "Insufficiency of the external recti in a case of convergent strabismus." Dr. LEONARD GUTHRIE, "Paralysis of ocular sympathetic."

July 14.—Clinical and pathological Meetings at Queen Square. Dr. W. J. POTTS, "A case of Friedreich's disease with onset late in life." Dr. FRED S. PALMER, "Friedreich's ataxia? (advanced stage), with an interesting family history." Dr. JAMES TAYLOR, "A case of Friedreich's disease with double optic atrophy." Dr. BUZZARD, "Disseminated sclerosis and functional paraplegia occurring in two sisters." Dr. BEEVOR, (1) "Pseudo bulbar paralysis." (2) "Myasthenia gravis." Dr. C. M. HINDS HOWELL, "Three cases illustrating the effects of cervical ribs." Dr. J. S. COLLIER, "Case of (?) vascular lesion of optic thalamus." Dr. JAMES TAYLOR, "Case in which symptoms of intracranial growth were relieved by trephining." Dr. G. A. SUTHERLAND, "Case in which symptoms of intracranial new growth were relieved by trephining." Dr. F. E. BATTEN, (*a*) "Case of brachial plexus lesion." (*b*) "Case of myopathy, exhibiting slow relaxation of muscles after muscular exertion." Dr. RAYNER BATTEN, "Congenital defect of left cervico-ocular sympathetic." Dr. S. A. K. WILSON, (1) "A case of spasmodic torticollis with involvement of the abdominal muscles"; (2) "A case of spasm of the

diaphragm"; (3) "A case of sensory aphasia." Under the care of Dr. FERRIER, shown by Dr. CARMALT JONES, "Myopathy associated with antecedent anterior poliomyelitis." Under the care of Dr. ORMEROD, shown by Dr. CARMALT JONES (*a*) "Lesions probably hæmorrhagic in lumbar cord; with intracranial arterial degeneration; (*b*) Left hemiplegia, lesion in right crus cerebri, also muscular wasting, and unusual respiratory asymmetry."

Pathological Specimens—Dr. PAGE MAY, Microscopical sections showing (*a*) Degeneration of Monakow's bundle. (*b*) Degeneration of vestibulo-spinal tract. (*c*) Degeneration of pre-dorsal bundle. Dr. LEONARD S. DUDGEON, Bacillus of Hamilton Wright isolated from a case of beri-beri. Dr. GORDON HOLMES, (1) Two complete cases of amyotrophic lateral sclerosis. (2) Syringomyelia with a spinal glioma. (3) Friedreich's ataxia (from one of Dr. Pott's series of cases). Dr. J. MICHELL CLARKE, Two specimens of cerebral tumour, illustrating the production of hemiplegia of apoplectic onset by hæmorrhage into a tumour; in one of the cases without any previous symptoms. Dr. C. E. BEEVOR, specimens of brains injected simultaneously with five colours.

November 1.—Ordinary Meeting. Presidential address by Sir THOMAS BARLOW, Bart., K.C.V.O., "Remarks on some of the nervous complications of specific fevers."

November 29.—"Hughlings Jackson Lecture," by Sir VICTOR HORSLEY, on "The illustration by recent research of Dr. HUGHLINGS JACKSON'S views on the functions of the cerebellum."

December 6.—Special Meeting to decide whether the Neurological Society should join in the proposed amalgamation of societies.

December 6.—Clinical Meeting, Dr. JAMES COLLIER, "A case of universal fixed and mobile spasm, with signs of pyramidal degeneration." Dr. T. D. SAVILL, "A case of clonic spasm in a girl, aged 17 years, for diagnosis." Dr. E. FARQUHAR BUZZARD, "A case for diagnosis." Dr. TRAVERS JONES (for Dr. WILFRED HARRIS), "Case of juvenile tabes showing a 'Charcot's joint.'" Dr. FREDERICK E. BATTEN, "A case of spinal new growth." Dr. FREDERICK S. PALMER, (*a*) "Double primary optic atrophy (complete)." (*b*) "Primary progressive myopathy (?)" Mr. DONALD ARMOUR, "Endothelioma of the left frontal region; removal by operation; recovery." Dr. WILFRED HARRIS, "Traumatic Sprengel shoulder." Dr. LEONARD GUTHRIE, "Poliomyelitis simulating Erb's paralysis from injury to fifth and sixth cervical nerves." Dr. S. A. K. WILSON, (1) "A case of torticollis in a child." (2) "A case of unilateral tremor." Dr. G. NEWTON PITT, "A case with violent ataxic movements of cerebellar type of uncertain origin." Dr. PARKES WEBER, "Hydrocephalus in an adult." Dr. T. D. SAVILL, "A case of chronic respiratory spasm in a boy, aged 17."

BALANCE SHEET. 1906.

Dr. NEUROLOGICAL SOCIETY. **Cr.**

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Secretaries' Petty Cash	1 15 11
Balance in Bank	212 0 11
	£505 2 8

Audited and found correct, January 30, 1907 — WILFRED HARRIS. T. GRAINGER STEWART.

RULES.

1.—The Society shall be called the NEUROLOGICAL SOCIETY OF THE UNITED KINGDOM.

2.—The objects of the Society shall be to promote the advance of Neurology and to facilitate intercourse amongst those who cultivate it, whether from a Psychological, Physiological, Anatomical, or Pathological point of view.

3.—The Society shall consist of Ordinary, Corresponding, and Honorary Members.

4.—Any one engaged in researches bearing on Neurology, or manifesting interest in such researches, shall be eligible for the Ordinary Membership.

5.—Men of distinction in Science, who have contributed to the advancement of Neurology, shall be eligible for the Honorary, or for the Corresponding Membership.

6.—The number of Honorary Members shall be limited to six, and that of Corresponding Members to twelve.

7.—Honorary and Corresponding Members shall have the right of attending the Meetings of the Society and of taking part in them.

8.—Honorary and Corresponding Members shall be elected by the Society on the recommendation of the Council.

9.—Candidates for Ordinary Membership shall be recommended by at least three Members of the Society, who shall append their names to a printed form supplied by the Secretaries to any person seeking Membership.

10.—The Council shall nominate candidates for election into the Society as Ordinary Members from among persons so recommended.

11.—The names of Candidates nominated by the Council shall be submitted to the next Meeting of the Society, and shall be balloted for at the next subsequent meeting, one black ball in ten excluding.

12.—The Annual Subscription of an Ordinary Member shall be One Guinea, due in advance.

13.—The Treasurer shall send to each Member, shortly before the Annual General Meeting, a notice reminding him that his Subscription becomes due on that day.

14.—Non-payment of the Subscription within twelve months after it is due shall be considered as equivalent to resignation.

15.—The Council shall consist of a President, two Vice-Presidents, two Secretaries, one Treasurer, the Editor of "Brain," and twelve Councillors, of whom not less than three shall be extra-Metropolitan Members.

16.—Five Members of the Council shall form a quorum.

17.—The office of President shall be tenable for one year, and be entered upon at the beginning of each year by the Senior Vice-President.

18.—The office of Vice-President shall be tenable for two years, one Vice-President being elected every year.

19.—The Secretaries and Treasurer shall be elected annually with eligibility for re-election.

20.—The Councillors shall be elected for one year, and not more than eight shall be eligible for re-election to the same office during the following year.

21.—One full week before the Annual General Meeting, the Secretaries shall send to each Member a balloting paper containing the names of the Officers and Councillors whom the Council nominate for the ensuing year.

22.—The Society shall elect the Council by ballot, each Member, however, being at liberty to substitute other names for any of those upon the list.

23.—There shall be six Ordinary Meetings annually, of which the first held in each year shall be the Annual General Meeting.

24.—Notices of each meeting, and of the subjects to be considered, shall be sent by the Secretaries to each Member of the Society at least one week before the meeting.

25.—Special Meetings shall be held at the option of the Council, or at the request, in writing, of twenty Members.

26.—The President shall have the power of inviting any person to attend and to take part in the scientific work of the meeting.

27.—If at any time the Council shall be of opinion that the interests of the Society require the expulsion of a Member, they shall submit the question to a special General Meeting, at which, if more than one-half of the Members of the Society vote, by ballot as usual, for the expulsion of the Member, his subscription for the current year shall be returned to him, and he shall thereupon cease to be a Member of the Society.

28.—The Council shall draw up and submit for the approval of the Society, supplementary rules regulating the dates, places, and character of the meetings; shall propose special subjects for investigation by the Society, and shall nominate sub-committees for the methodical carrying out of such investigations. They shall decide, from time to time, on the form of publication which its proceedings are to assume.

29.—No alteration shall be made in the present rules, excepting at the Annual Meeting, or at a Special Meeting convened for the purpose, and unless it be proposed by the Council, or in writing by at least twenty Members, the usual notice be given of the proposed change to every Member before the meeting at which it is to be brought forward.

Each Member receives quarterly, from the beginning of the year in which he is elected, a copy of "BRAIN; a Journal of Neurology," the organ of the Society, and edited for it by Henry Head, M.D., F.R.S.

HONORARY MEMBERS.

Professor WUNDT, Leipzig.
,, HITZIG, Halle.
Sir SAMUEL WILKS, Bart., London.
Dr. J. HUGHLINGS JACKSON, London.

CORRESPONDING MEMBERS.

Professor DEJERINE, Paris.
,, ERB, Heidelberg.
,, GOLGI, Pavia.
,, GRASSET, Montpellier.
,, LUCIANI, Rome.
,, MUNK, Berlin.
,, OBERSTEINER, Vienna.
,, A. PICK, Prague.
,, RETZIUS, GUSTAF, Stockholm.
,, RIBOT, Paris.
Dr. WEIR MITCHELL, Philadelphia.
Dr. ALLEN STARR, New York.

LIST OF PRESIDENTS FROM THE FOUNDATION OF THE SOCIETY.

1886 J. HUGHLINGS JACKSON, M.D., F.R.S.
1887 Sir SAMUEL WILKS, Bart., M.D., F.R.S.
1888 Sir JAMES CRICHTON-BROWNE, M.D., F.R.S.
1889 JONATHAN HUTCHINSON, F.R.S.
1890 THOMAS BUZZARD, M.D.
1891 JOHN S. BRISTOWE, M.D., F.R.S.
1892 H. CHARLTON BASTIAN, M.D., F.R.S.
1893 EDWARD A. SCHÄFER, F.R.S.
1894 DAVID FERRIER, M.D., F.R.S.
1895 Sir WILLIAM H. BROADBENT, Bart., M.D., F.R.S.
1896 ALEXANDER HILL, M.A., M.D.
1897 GEORGE H. SAVAGE, M.D.
1898 Sir VICTOR HORSLEY, M.B., F.R.C.S., F.R.S.
1899 W. H. GASKELL, M.D., F.R.S.
1900 AUGUSTUS WALLER, M.D., F.R.S.
1901 W. JULIUS MICKLE, M.D.
1902 HERBERT WILLIAM PAGE, M.A., F.R.C.S.
1903 J. N. LANGLEY, D.Sc., F.R.S.
1904 S. J. SHARKEY, M.D.
1905 Sir JOHN BATTY TUKE, M.D., LL.D.
1906 Sir THOMAS BARLOW, Bart., K.C.V.O., M.D., F.R.C.P.

LIST OF HUGHLINGS JACKSON LECTURERS.

1897 J. HUGHLINGS JACKSON, M.D., F.R.S.
1900 Geheimrath Professor HITZIG, Halle.
1903 Sir WILLIAM H. BROADBENT, Bart., M.D., F.R.S.
1906 Sir VICTOR HORSLEY, M.B., F.R.C.S., F.R.S.

*** Members are requested to communicate with the Secretaries when corrections are necessary.

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EXPLANATION OF ABBREVIATIONS.

O.M., Original Member.	Tr., Treasurer.
Pres., President.	Sec., Secretary.
V.-P., Vice-President.	C., Member of Council.

H.J.L., Hughlings Jackson Lecturer.

ORDINARY MEMBERS.

Elected

1899 ABRAHAMS, BERTRAM, M.B., F.R.C.P., 14, Welbeck Street, W.

1897 ANDERSON, H. KERR, M.D., B.Sc., Whitefield, Great Shelford, Cambs.

1905 ARKWRIGHT, J. A., M.D., 13, Welbeck Street, W.

1903 ARMOUR, D. J., M.B., F.R.C.S., 89, Harley Street, W.

1904 ASCHERSON, W. LAWRENCE, M.D., M.R.C.P., 3, Whitehall Court, S.W.

1891 BALLANCE, C. A., M.S., F.R.C.S., 106, Harley Street, W. (C., 1897-1900.)

O.M. BARLOW, Sir THOMAS, Bart., K.C.V.O., M.D., F.R.C.P., 10, Wimpole Street, W. (Pres., 1906; V.-P., 1904-05; C., 1907.)

1902 BARNES, A. STANLEY, M.D., M.R.C.P., 141, Great Charles Street, Birmingham.

O.M. BASTIAN, H. CHARLTON, M.D., F.R.C.P., F.R.S., 8A, Manchester Square, W. (Pres., 1892; V.-P., 1891; C., 1886-89, 1893-95.)

1897 BATTEN, FREDERICK E., M.D., F.R.C.P., 93, Harley Street, W. (Sec., 1903-06; C., 1907.)

1903 BAYLISS, W. M., M.A., D.Sc., F.R.S., St. Cuthbert's, West Heath Road, Hampstead.

O.M. BEACH, FLETCHER, M.B., F.R.C.P., Winchester House, Kingston Hill, Surrey. (C., 1903-06.)

1903 BEDDARD, A. P., M.D., F.R.C.P., 44, Seymour Street, Portman Square.

O.M. BEEVOR, C. E., M.D., F.R.C.P., 135, Harley Street, W. (Tr., 1894-1905; V.-P., 1905-06; C., 1893; Pres., 1907.)

1903 BERRY, GEORGE A., M.B., F.R.C.S.Edin., 31, Drumsheugh Gardens, Edinburgh.

1898 BOLTON, JOSEPH SHAW, M.D., B.S., B.Sc., County Asylum, Rainhill, near Liverpool.

1896 BOND, C. HUBERT, M.D., D.Sc., London County Colony for Epileptics, Ewell, Surrey.

1899 BOND, J. W., M.D., B.S., 26, Harley Street, W.

1892 BOWLBY, ANTHONY A., C.M.G., F.R.C.S., 24, Manchester Square, W.

1898 BOYCE, RUBERT W., M.B., F.R.S., University College, Liverpool.

1889 BRADFORD, J. R., M.D., D.Sc., F.R.C.P., F.R.S., 8, Manchester Square, W.

Elected

O.M. BRAMWELL, BYROM, M.D., F.R.C.P.Ed., 23, Drumsheugh Gardens, Edinburgh. (C., 1894, 1903-05.)

1899 BRAMWELL, EDWIN, M.B., C.M., M.R.C.P., 23, Drumsheugh Gardens, Edinburgh.

1895 BRAMWELL, JOHN MILNE, M.B., C.M., 33, Wimpole Street, W.

1892 BRISTOWE, HUBERT CARPENTER, M.D., Wrington, Somerset.

O.M. BROADBENT, Sir WM. H., Bart., K.C.V.O., M.D., F.R.C.P., F.R.S., 84, Brook Street, W. (Pres., 1895; V.-P., 1893; C., 1886-87, 1896.)

1894 BRODIE, T. GRIGOR, M.D., F.R.S., 4, Lancaster Terrace, N.W.

1897 BROWN, H. H., M.D., F.R.C.S., 22, Museum Street, Ipswich.

1888 BROWN, SANGER, M.D., Kenilworth, Chicago, Ill., U.S.A.

O.M. BRUCE, ALEX., M.D., F.R.C.P.Ed., 8, Ainslie Place, Edinburgh. (C., 1895.)

O.M. BRUCE, J. MITCHELL, M.D., F.R.C.P., 23, Harley Street, W.

O.M. BRUNTON, Sir T. LAUDER, M.D., F.R.C.P., F.R.S., 10, Stratford Place, W. (C., 1896-98.)

1895 BRYAN, FREDERICK, M.B., 41, Welbeck Street, W.

1903 BUBB, WILLIAM, M.R.C.S., L.R.C.P., County Asylum, Powick, Worcester.

1890 BURY, JUDSON SYKES, M.D., F.R.C.P., 10, St. John Street, Manchester. (C. 1905-06.)

1900 BUZZARD, E. FARQUHAR, M.D., F.R.C.P., 33, Harley Street, W. (Sec., 1907.)

O.M. BUZZARD, THOMAS, M.D., F.R.C.P., 74, Grosvenor Street, W. (Pres., 1890; V.-P., 1888-89; C., 1886-87, 1891-95.)

1898 CALEY, H. A., M.D., F.R.C.P., 24, Upper Berkeley Street, W.

1903 CAMPBELL, A. W., M.D., General Post Office, Sydney, New South Wales.

1898 CAMPBELL, HARRY, M.D., F.R.C.P., 23, Wimpole Street, W.

1906 CARMALT-JONES, D. W., M.B., M.R.C.P., 120, Maida Vale, W.

1899 CHAMBERS, JAMES, M.A., M.D., The Priory, Roehampton.

1891 CLARKE, J. MICHELL, M.D., F.R.C.P., 28, Pembroke Road, Clifton, Bristol. (C., 1903-05.)

1903 CLOUSTON, T. S., M.D., F.R.C.P.Ed., Royal Asylum, Morningside, Edinburgh.

1892 COLE, ROBERT HENRY, M.D., M.R.C.P., 48, Upper Berkeley Street, Portman Square.

1900 COLE, SYDNEY JOHN, M.D., Wilts County Asylum, Devizes.

1899 COLLIER, J. S., M.D., B.Sc., F.R.C.P., 57A, Wimpole Street, W. (C., 1905-06.)

1890 COLMAN, W. S., M.D., F.R.C.P., 9, Wimpole Street, W. (Sec., 1899-1902. C., 1903-05.)

1890 CORNER, HARRY, M.D., Brook House, Southgate, Middlesex.

1899 COUPLAND, SIDNEY, M.D., F.R.C.P., 16, Queen Anne Street, W.

1901 COX, JOSHUA JOHN, M.D., St. Ronan's, Clarendon Road, Eccles, Manchester.

1901 CRAIG, MAURICE, M.D., F.R.C.P., Bethlem Royal Hospital, S.E. (C., 1907.)

1905 CUNNINGHAM, D. J., M.D., 18, Grosvenor Crescent, Edinburgh.

1898 DARLEY-HARTLEY, W., M.D., M.R.C.P., Main Road, Sea Point, Cape Town, South Africa.

1895 DAVIDSON, ANDREW, M.D., Callan Park, Sydney, N.S.W.

Elected

1897 DAWSON, WILLIAM R., M.D., F.R.C.P.I., Farnham House, Finglas, co. Dublin, Ireland.

1898 DODD, HENRY WORK, F.R.C.S., 136, Harley Street, W.

O.M. DRESCHFELD, JULIUS, M.D., F.R.C.P., Stanley House, Wilmslow Road, Withington, Manchester. (V.-P., 1898-99.)

O.M. DRUMMOND, DAVID, M.D., 6, Saville Place, Newcastle-on-Tyne.

1903 DUDGEON, L. S., M.R.C.P., 6, Powis Gardens, London, W.

1892 DUPUY, EUGENE, M.D., 53, Avenue Montaigne, Paris.

1906 EWENS, GEO. FRANCIS, M.D., c/o Messrs. Grindley, 54, Parliament Street, S.W.

1899 FAWCETT, JOHN, M.D., F.R.C.P., 66, Wimpole Street, W.

O.M. FERRIER, DAVID, M.D., F.R.C.P., F.R.S., 34, Cavendish Square, W. (Pres., 1886; V.-P., 1892-3; C., 1886, 1890-95.)

1903 FLASHMAN, J. F., M.B., Ch.M., B.Sc., Totnes, Temple Street, Petersham, Sydney, Australia.

1903 FLEMING, ROBERT, M.D., F.R.C.P.Ed., 10, Chester Street, Edinburgh.

1903 FLETCHER, H. MORLEY, M.D., F.R.C.P., 98, Harley Street, W.

O.M. GASKELL, W. H., M.D., F.R.S., The Uplands, Great Shelford, Cambs. (Pres., 1899; V.-P., 1897-98; C., 1896, 1900.)

1903 GIBSON, G. A., M.D., D.Sc., F.R.C.P.Ed., 3, Drumsheugh Gardens, Edinburgh.

1904 GIGLIOLI GUIOLO T., M.D., M.R.C.P., Via Del Campidoglio, 2, Florence.

O.M. GODLEE, R. J., M.B., F.R.C.S., 19, Wimpole Street, W.

1905 GOLLA, F. LUCIEN, M.B., B.Ch., M.R.C.P., 149, St. James' Court, S.W.

1899 GOODALL, EDWIN, M.D., F.R.C.P., Joint Counties Asylum, Carmarthen.

1897 GOSSAGE, ALFRED MILNE, M.B., F.R.C.P., 54, Upper Berkeley Street, W.

1892 GOTCH, FRANCIS, M.A., F.R.S., The Lawn, Banbury Road, Oxford.

1893 GRANT, J. DUNDAS, M.D., F.R.C.S., 18, Cavendish Square, W.

1906 GRÜNBAUM, OTTO, M.D., M.R.C.P., 34, Wimpole Street, W.

O.M. GUNN, R. MARCUS, M.B., F.R.C.S., 54, Queen Anne Street, W. (C., 1899-1900.)

1895 GUTHRIE, LEONARD, M.D., F.R.C.P., 15, Upper Berkeley Street, W. (C., 1904; Sec. 1905-06.)

1906 HALL, GEORGE, M.D., M.R.C.P., National Hospital, Queen Square, W.C.

1902 HALL, W. HAMILTON, M.R.C.S., Fordcombe, Tunbridge Wells.

1893 HALLIBURTON, WILLIAM DOBINSON, M.D., F.R.C.P., F.R.S., Church Cottage, 17, Marylebone Road, N.W.

O.M. HAMILTON, D. J., M.B., F.R.S.E., University, Aberdeen.

1898 HANBURY, WILLIAM R., M.R.C.S., L.R.C.P., West Ham Borough Asylum, Goodmayes, Ilford.

1904 HARRIS, DAVID, F., M.D., The University, St. Andrews.

1897 HARRIS, WILFRED J., M.D., F.R.C.P., 61, Wimpole Street, W. (C., 1907.)

1906 HART, BERNARD, M.B., Herts County Asylum, Hill End, St. Albans.

1891 HAWKINS, HERBERT PENNELL, M.D., F.R.C.P., 56, Portland Place, W.

O.M. HAYCRAFT, J. B., M.D., D.Sc., F.R.S.E., University College of South Wales, Cardiff.

Elected

1891 HEAD, HENRY, M.D., F.R.C.P., F.R.S., 4, Montagu Square, Hyde Park, W. (C., 1900; Sec., 1901-1904; Editor of "Brain," 1905-06.)

O.M. HILL, ALEX., M.A., M.D., Downing Lodge, Cambridge. (Pres., 1896; V.-P., 1895; C., 1892-94.)

1905 HOBHOUSE, EDMUND, M.D., F.R.C.P., 12, Second Avenue, Brighton (Hove).

1903 HOLMES, GORDON M., M.D., B.Ch., National Hospital, Queen Square, W.C.

O.M. HORSLEY, Sir VICTOR, F.R.C.S., F.R.S., 25, Cavendish Square, W. (Pres., 1898; V.-P., 1896-97; C., 1892-93, 1899; H.J.L., 1906.)

1903 HOWLAND, GOLDWIN, W., M.B., M.R.C.P., 540, Spadina Avenue, Toronto, Canada.

1905 HOWELL, C. M. HINDS, M.B., B.Ch., M.R.C.P., 53, Queen Anne Street, W.

1889 HUGGARD, WILLIAM R., M.D., F.R.C.P., Davoz Platz, Switzerland.

1896 HULME, GEORGE FREDERICK, M.B., C.M., South View, Montague Road, Felixstowe.

1904 HUMPHREYS, F. R., M.R.C.S., L.R.C.P., 2, Chalcot Gardens, England Lane, N.W.

1889 HUMPHRY, LAURENCE, M.D., F.R.C.P., Lensfield, Cambridge.

1903 HUNTER, WALTER K., M.D., D.Sc., 7, Woodside Place, Glasgow.

1901 HUNTER, WILLIAM, M.B., C.M., Government Bacteriological Laboratory, Hong Kong.

1903 HUNTER, WILLIAM, M.D., F.R.C.P., 103, Harley Street, W.

O.M. HUTCHINSON, JONATHAN, F.R.C.S., F.R.S., 15, Cavendish Square, W. (Pres., 1889; V.-P., 1887-88; C., 1886, 1890.)

1889 HYSLOP, T. B., M.D., C.M., M.R.C.P.Ed., Bethlem Royal Hospital, Lambeth, S.E.

O.M. JACKSON, J. HUGHLINGS, M.D., F.R.C.P., F.R.S., 3, Manchester Square, W. (Pres., 1886; H.J.L., 1897; C., 1887, 1892.) Hon. Memb., 1904.

O.M. JESSOP, W. H., M.B., F.R.C.S., 73, Harley Street, W.

1895 JOHNSTON, GEORGE, M.D., M.R.C.P., 3, Montagu Place, Montagu Square, W.

1903 JONES, A. E., M.B., B.Sc., M.R.C.P., 13, Harley Street, W.

1892 JONES, HENRY LEWIS, M.D., F.R.C.P., 143, Harley Street, W.

1894 JONES, ROBERT, M.D., F.R.C.S., Claybury Asylum, Woodford, Essex.

1904 JONES, ROBERT, F.R.C.S.Ed., 11, Nelson Street, Liverpool.

1902 KELYNACK, T. N., M.D., M.R.C.P., 120, Harley Street, W.

1894 KIDD, PERCY, M.D., F.R.C.P., 60, Brook Street, W. (C., 1904-06.)

1906 KNAPP, PHILIP COOMBS, M.D., 535, Beacon Street, Boston.

1896 LANGDON, F. W., M.D., Cincinnati, Ohio, U.S.A.

1898 LANGDON-DOWN, REGINALD L., M.B., M.R.C.P., 47, Welbeck Street, W.

O.M. LANGLEY, J. N., M.A., D.Sc., F.R.S., Hedgerley Lodge, Madingley Road, Cambridge. (Pres., 1903; V.-P., 1901-1902; C., 1898-99, 1904.)

1888 LAWFORD, J. B., M.D., F.R.C.S., 99, Harley Street, W. (C., 1902-3.)

O.M. LEES, D. B., M.D., F.R.C.P., 22, Weymouth Street, W. (C., 1901-1903.)

1897 LEWIS, H. WOLSELEY, M.R.C.S., L.R.C.P., London County Asylum, Banstead, Surrey.

Elected

1904 LINDSAY, J. A., M.D., F.R.C.P., 13, College Square E., Belfast.
1897 LOEWENTHAL, MAX, M.D., M.R.C.P., 126, Princes' Road, Liverpool.
1899 LORD, JOHN R., M.B., C.M., London County Asylum, Bexley, Kent.

1898 MCALDOWIE, ALEXANDER M., M.D., 6, Brook Street, Stoke-on-Trent.
1898 MACCORMAC, JOHN M'GEE, M.D., 73, Victoria Place, Belfast.
1891 MACDONALD, PETER WILLIAM, M.D., C.M., The County Asylum, Dorchester, Dorset.
1902 MCDOUGALL, WM., M.B., Woodsend, Foxcombe Hill, nr. Oxford.
1906 MACKAY, H. J., M.D., Lansdowne House, Devizes.
1902 MACKENZIE, JAMES, M.D., Bank Parade, Burnley.
O.M. MACKENZIE, Sir STEPHEN, M.D., F.R.C.P., 31, Cavendish Square, W. (C., 1896-98.)
1896 MACKINTOSH, ASHLEY W., M.A., M.D., 9, Bon Accord Square, Aberdeen.
1903 MACNAMARA, E. D., M.B., B.C., M.R.C.P., 9, Welbeck Street, W.
O.M. MAGUIRE, ROBERT, M.D., F.R.C.P., 4, Seymour Street, W. (Sec., 1890-93; C., 1893-96.)
O.M. MANN, J. DIXON, M.D., F.R.C.P., 16, St. John Street, Manchester.
1902 MANTLE, ALFRED, M.D., M.R.C.P., Savile Place, Halifax.
1901 MARRIS, W. A., M.D., Springfield Road, King's Heath, Birmingham.
1894 MARTIN, SIDNEY, M.D., F.R.C.P., F.R.S., 10, Mansfield Street, W.
1905 MAVROGORDATO, A., M.R.C.S., L.R.C.P., St. Thomas' Hospital, S.E.
1891 MAY, W. PAGE, M.D., F.R.C.P., Physiological Lab., University College, London (C., 1906.)
1906 MAYNARD, E. F., M.D., M.R.C.P., 49, Brunswick Place, Hove.
1890 MENZIES, WILLIAM FRANCIS, M.D., B.Sc., M.R.C.P., Cheddleton Asylum, Leek, Staffordshire.
O.M. MERCIER, C., M.B., F.R.C.P., 34, Wimpole Street (C., 1899-1902.)
1897 MEYER, ADOLF, M.D., LL.D., Pathological Institute, New York State Hospital, Ward's Island, N.Y., U.S.A.
O.M. MICKLE, W. JULIUS, M.D., F.R.C.P., Grove Hall Asylum, Bow, E. (Pres., 1901; V.-P., 1900; C., 1895-97, 1902-4.)
1895 MOORE, NORMAN, M.D., F.R.C.P., 94, Gloucester Place, W.
O.M. MORRIS, MALCOLM, F.R.C.S.Ed., 8, Harley Street, W.
1902 MORRISON, C. S., L.R.C.P.E., County and City Asylum, Hereford.
1888 MOTT, F. W., M.D., F.R.C.P., F.R.S., 25, Nottingham Place, W. (Sec., 1894-98; C., 1899-1902.)
1901 MOULD, GEORGE WM., M.R.C.S., Oakhurst, Colwyn Bay.
1899 MOULD, GILBERT E., M.R.C.S., L.R.C.P., The Grange, nr. Rotherham.
1894 MURRAY, GEORGE R., M.A., M.D., F.R.C.P., 11, Ellison Place, Newcastle-on-Tyne (C., 1906.)
1889 MURRAY, H. MONTAGUE, M.D., F.R.C.P., 25, Manchester Square, W.
1904 MYERS, CHARLES S., M.D., Melrose, Grange Road, Cambridge.

1904 NEILD, NEWMAN, M.D., Richmond Hill, Clifton, Bristol.
O.M. NETTLESHIP, EDWARD, F.R.C.S., Shottermill, Haslemere, Surrey.
1902 NEWINGTON, HAYES, F.R.C.P.Ed., The Gables, Ticehurst, Sussex.
O.M. NICOLSON, DAVID, C.B., M.D., M.R.C.P.Ed., Balgownie, Guildford, Surrey.
1888 NIERMEYER, J. H. H., M.D., Amsterdam.

Elected.

1904 NOLAN, M. J., L.R.C.P., L.R.C.S.I., Down District Asylum, Downpatrick.

1888 NORMAN, CONOLLY, F.R.C.P.I., F.R.C.S.I., St. Dymphna's, N. Circular Road, Dublin.

1887 OGILVIE, GEORGE, M.B., M.R.C.P., 22, Welbeck Street, W.

1887 OLIVER, THOMAS, M.D., F.R.C.P., 7, Ellison Place, Newcastle-on-Tyne.

O.M. ORANGE, WM., C.B., M.D., F.R.C.P., Oakhurst, Godalming, Surrey.

O.M. ORMEROD, J. A., M.D., F.R.C.P., 25, Upper Wimpole Street, W. (C., 1895-97; V.-P., 1907.)

1899 ORR, DAVID, M.D., County Asylum, Prestwich, Manchester.

1904 OWEN, ARTHUR GEOFFRY, M.D., B.Sc., Terang, Victoria, Australia.

1905 OWEN, S. A., M.B., B.C., 50, Harrington Gardens, Kensington, S.W.

O.M. PAGE, HERBERT WILLIAM, M.A., F.R.C.S., 146, Harley Street, W. (Pres., 1902; V.-P., 1900-1; C., 1891-92, 1903-06.)

1903 PALMER, FREDK. S., M.D., M.R.C.P., 19, Harley Street, W.

1895 PARKINSON, JOHN PORTER, M.D., M.R.C.P., 57, Wimpole Street, W.

1901 PARSONS, JOHN HERBERT, D.Sc., F.R.C.S., 27, Wimpole Street, W.

1901 PASSMORE, E. S., M.D., M.R.C.P., Croydon Asylum, Warlingham, Surrey.

1904 PATON, LESLIE, J., M.B., F.R.C.S., 1, Spanish Place, W.

1905 PIERCE, BEDFORD, M.D., F.R.C.P., The Retreat, York.

1899 PIPER, FRANCIS P., M.B., M.R.C.S., L.R.C.P., King's Leigh, Tankerton, Whitstable.

1888 PITT, G. N., M.D., F.R.C.P., 15, Portland Place, W. (C., 1903-05.)

1899 RAMBAUT, DANIEL F., M.D., B.Ch., County Asylum, Bicton Heath, Shrewsbury.

1894 RANSOM, WILLIAM B., M.D., F.R.C.P., The Pavement, Nottingham.

1890 RAYNER, HENRY, M.D., M.R.C.P.Ed., 16, Queen Anne Street, W.

O.M. REID, E. W., M.B., F.R.S., University College, Dundee.

1897 REISSMANN, CHARLES H., M.B., B.Sc., St. Peter's, Adelaide, South Australia.

1893 RENNIE, GEORGE E., M.D., M.R.C.P., College Street, Sydney.

1895 REYNOLDS, ERNEST S., M.D., F.R.C.P., 2, St. Peter's Square, Manchester.

1906 RICHARDS, JOHN, M.B., C.M., F.R.C.S., County Asylum, Leicester.

1891 RIVERS, W. H. R., M.A., M.D., F.R.C.P., St. John's College, Cambridge. (C., 1897-98.)

1905 ROBERTSON-MILNE, CHAS. JOHN, I.M.S., M.B., C.M., Superintendent, Punjab Asylum, Lahore.

1893 ROBERTSON, WILLIAM FORD, M.D., 10, Morningside Terrace, Edinburgh.

1892 ROWE, EDMUND LEWIS, L.R.C.P., L.R.C.S., Borough Asylum, Ipswich.

1901 ROWS, R. G., M.D., County Lunatic Asylum, Lancaster.

1892 RUFFER, MARC ARMAND, M.D., Ramleh, Egypt.

1905 RUSSEL, COLIN K., M.D., C.M., 51, Mackay Street, Montreal, Canada.

1903 RUSSELL, A. E., M.D., M.R.C.P., 9, Wimpole Street, W.

1889 RUSSELL, J. S. RISIEN, M.D., F.R.C.P., 44, Wimpole Street, W. (Sec., 1896-1900; C., 1901-1904.)

1899 RUSSELL, J. W., M.D., M.R.C.P., 72, Newhall Street, Birmingham.

Elected

1901 SALAMAN, REDCLIFFE N., M.A., M.B., Frognal End, Frognal Gardens, Hampstead.

1906 SARGENT, PERCY W., F.R.C.S., 67A, Harley Street, W.

O.M. SAVAGE, G. H., M.D., F.R.C.P., 3, Henrietta Street, Cavendish Square. (Pres., 1897; V.-P., 1895; C., 1886-90, 1898.)

1888 SAVILL, T. D., M.D., M.R.C.P., 60, Upper Berkeley Street, W.

O.M. SCHÄFER, E. A., F.R.S., University of Edinburgh. (Pres., 1893; V.-P., 1892; C., 1886-98.)

O.M. SEMON, Sir FELIX, C.V.O., M.D., F.R.C.P., 39, Wimpole Street, W. (C., 1899-1901.)

1898 SEWARD, WM. JOSEPH, M.B., Colney Hatch Asylum, N.

O.M. SHARKEY, S. J., M.D., F.R.C.P., 22, Harley Street, W. (Pres., 1904; V.-P., 1902-3; C., 1899-92, 1905.)

O.M. SHERRINGTON, C. S., M.D., F.R.S., 16, Grove Park, Liverpool. (V.-P., 1906; C., 1892-95.)

1894 SHUTTLEWORTH, GEORGE E., M.D., Parkholme, East Sheen, S.W.

1900 SINGER, H. D., M.D., M.R.C.P., Maccagno Buildings, Omaha, Neb., U.S.A.

1895 SMITH, F. J., M.D., F.R.C.P., 138, Harley Street, W.

1887 SMITH, R. PERCY, M.D., F.R.C.P., 36, Queen Anne Street, W. (Editor of "Brain," 1901-1904; C. 1905-06.)

1895 SPICER, W. T. HOLMES, M.B., F.R.C.S., 5, Wimpole Street, W.

1895 STANLEY, DOUGLAS, M.D., M.R.C.P., 21, Broad Street, Birmingham.

1895 STANSFIELD, THOS. E. K., M.B., London County Asylum, Bexley, Kent.

1892 STARLING, ERNEST HENRY, M.D., F.R.C.P., F.R.S., 40, West End Lane, Hampstead (C., 1904-06.)

1901 STEELL, GRAHAM, M.D., F.R.C.P., 96, Mosley Street, Manchester.

1897 STEWART, PURVES, M.A., M.D., M.R.C.P., 7, Harley Street, W.

1903 STEWART, T. GRAINGER, M.B., B.Ch., M.R.C.P., 26, Queen Anne Street, W.

1897 STIRLING, WM., M.D., D.Sc., Owens College, Manchester.

1897 STODDART, WM. H., M.D., M.R.C.P., Bethlem Royal Hospital, Lambeth, S.E.

1902 SULLIVAN, WM. CHARLES, M.D., H.M. Prison, Pentonville, N.

1904 SUTHERLAND, GEO. ALEXANDER, M.D., F.R.C.P., 73, Wimpole Street, W.

1898 TAYLOR, F. R. P., M.D., East Sussex Asylum, Hellingly, Sussex.

1891 TAYLOR, JAMES, M.D., F.R.C.P., 49, Welbeck Street, W. (C., 1900-03; Tr., 1905-06.)

1907 THOMPSON, H. THEODORE, M.D., F.R.C.S., M.R.C.P., 35, Harley Street, W.

1898 THOMSON, H. CAMPBELL, M.D., F.R.C.P., 31, Queen Anne Street, W.

1889 THORBURN, WM., F.R.C.S., Oxford Lodge, Victoria Park, Manchester. (C., 1901-1903.)

1892 TITCHENER, E. B., B.A., Ph.D., Cornell Heights, Ithaca, N.Y.

O.M. TOOTH, H. H., C.M.G., M.D., F.R.C.P., 34, Harley Street, W. (Sec., 1891-95; C., 1896-99.)

1892 TREVELYAN, E. F., M.D., B.Sc., F.R.C.P., 40, Park Square, Leeds.

1892 TUCKEY, CHARLES LLOYD, M.D., 88, Park Street, Grosvenor Square, W.

1892 TUKE, Sir J. BATTY, M.D., F.R.C.P.Ed., F.R.S.Ed., 20, Charlotte Square, Edinburgh. (Pres., 1905; V.-P., 1903-04.)

Elected

1891 TUKE, THOMAS SEYMOUR, M.B., Chiswick House, Chiswick.

1891 TURNER, WILLIAM ALDREN, M.D., F.R.C.P., 18, Harley Street. (C. 1906.)

1898 TURNEY, HORACE G., M.D., F.R.C.P., 68, Portland Place, W. (C., 1906.)

O.M. TWEEDY, Sir JOHN, F.R.C.S., 100, Harley Street, W.

1901 VINCENT, SWALE, M.B., Physiological Laboratory, University of Manitoba, Winnipeg, Canada.

1888 VOORTHUS, J. A., M.D., Apeldoorn, 542, Loulaan, Netherlands.

O.M. WALLER, AUGUSTUS, M.D., F.R.S., 32, Grove End Road, N.W. (Pres., 1900; V.-P., 1899; C., 1894-97, 1901.)

O.M. WARNER, FRANCIS, M.D., F.R.C.P., 5, Prince of Wales Terrace, Kensington Palace, W.

1902 WARRINGTON, W. B., M.D., F.R.C.P., 69, Rodney Street, Liverpool. (C., 1907.)

1904 WATSON, CHALMERS, M.B., F.R.C.P.E., 22, Coates Crescent, Edinburgh.

O.M. WATTEVILLE, A. DE, M.A., M.D., B.Sc., Rolle, Switzerland (C., 1890-95, 1896-1902; Sec., 1886-89; Editor of "Brain," 1880-1900.)

1897 WEBER, F. PARKES, M.D., F.R.C.P., 19, Harley Street, W.

1897 WEST, SAMUEL, M.D., F.R.C.P., 15, Wimpole Street, W.

O.M. WHITE, W. HALE, M.D., F.R.C.P., 65, Harley St., W. (C., 1897-1902.)

1894 WHITING, ARTHUR J., M.D., M.R.C.P., 142, Harley Street, W.

1889 WIGLESWORTH, JOSEPH, M.D., F.R.C.P., County Asylum, Rainhill, Lancashire.

1905 WILKIN, GRIFFITH CHARLES, M.R.C.S., L.R.C.P.Lond., Conway House, Paignton, Devon.

1900 WILLIAMS, LEONARD, M.D., M.R.C.P., 8, York Street, Portman Square, W.

1894 WILLIAMSON, RICHARD T., M.D., F.R.C.P., 2, St. Peter's Square, Manchester.

1896 WILSON, ALBERT, M.D., 1, Belsize Park, N.W.

1903 WILSON, G. R., M.D., M.R.C.P.Ed., 8, Rutland Square, Edinburgh.

1905 WILSON, S. A. K., M.A., M.B., Ch.B., B.Sc., National Hospital, Queen Square, Bloomsbury.

1896 WOOD, GUY M., M.B., 49, Gordon Square, W.C.

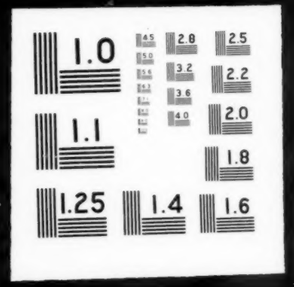
1889 WOOD, T. OUTTERSON, M.D., F.R.C.P.Ed., 40, Margaret Street, Cavendish Square, W.

1892 WOODHEAD, G. SIMS, M.D., F.R.C.P.Ed., 6, Scrope Terrace, Cambridge.

1899 WRIGHT, HAMILTON, M.D., London School of Tropical Medicine.

1898 YOUNG, ROBERT ARTHUR, M.D., B.Sc., F.R.C.P., 41, Wimpole St., W.

RESOLUTION CHART



100 MILLIMETERS

INSTRUCTIONS Resolution is expressed in terms of the lines per millimeter recorded by a particular film under specified conditions. Numerals in chart indicate the number of lines per millimeter in adjacent "T-shaped" groupings.

In microfilming, it is necessary to determine the reduction ratio and multiply the number of lines in the chart by this value to find the number of lines recorded by the film. As an aid in determining the reduction ratio, the line above is 100 millimeters in length. Measuring this line in the film image and dividing the length into 100 gives the reduction ratio. Example: the line is 20 mm. long in the film image, and $100/20 = 5$.

Examine "T-shaped" line groupings in the film with microscope, and note the number adjacent to finest lines recorded sharply and distinctly. Multiply this number by the reduction factor to obtain resolving power in lines per millimeter. Example: 7.9 group of lines is clearly recorded while lines in the 10.0 group are not distinctly separated. Reduction ratio is 5, and $7.9 \times 5 = 39.5$ lines per millimeter recorded satisfactorily. $10.0 \times 5 = 50$ lines per millimeter which are not recorded satisfactorily. Under the particular conditions, maximum resolution is between 39.5 and 50 lines per millimeter.

Resolution, as measured on the film, is a test of the entire photographic system, including lens, exposure, processing, and other factors. These rarely utilize maximum resolution of the film. Vibrations during exposure, lack of critical focus, and exposures yielding very dense negatives are to be avoided.